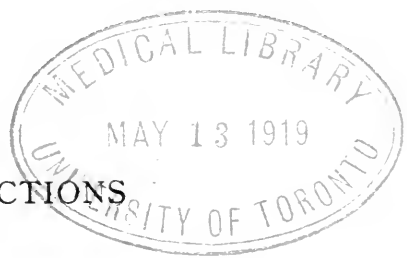


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TRANSACTIONS

OF THE

TWENTY-SECOND AND TWENTY-THIRD
ANNUAL MEETINGS

OF THE

American
Academy of Ophthalmology
and Oto-Laryngology

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HELD AT

PITTSBURGH, PA., OCTOBER 29 AND 30, 1917

AND

DENVER, COLO., AUGUST 5 AND 6, 1918

This volume contains the papers read at the Twenty-second Annual Meeting held at Pittsburgh, Pa., October 29 and 30, 1917, and those read at the Twenty-third Annual Meeting held at Denver, Colo., August 5 and 6, 1918.

The Twenty-fourth Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology will be held at Cleveland, Ohio.

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| BALLENGER, WM. L., Chicago, Ill..... | 1903 |
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| BROWN, JOHN E., Columbus, Ohio..... | 1916 |
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| INGERSOLL, J. M., Cleveland, Ohio..... | 1919 |

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PRESIDENT'S ADDRESS

W. L. DAYTON, M.D.

LINCOLN, NEB.

It has been the custom in all well regulated societies, for the president, at the opening of each annual session, to address the members on some subject, scientific or otherwise. This society, being a well regulated one, has established this precedent. I, therefore, as your president, must comply. While this custom demands my compliance, I find nothing in precedent nor in the Constitution or By-Laws that compels, you, as Fellows, to remain for the ordeal.

As to the subject of this address, some might be so impolitic or impolite as to suggest it was the ramblings of an old man. Here, however, I take issue with you. Having grown up with this society, seen its strong, vigorous growth, partaken of the intellectual feasts, and watched the influx of brilliant young men, I have been rejuvenated, and am, today, a vigorous young-old man.

Due consideration must be given to the fact that in the earlier days of this Academy the triumvirate of specialists in medicine and surgery represented by the Fellows of this society were few, confined largely, as it were, to medical centers and to the larger cities. The opportunity for study along these special lines was limited. There was a time within the memory of a number of our members when it was quite impossible to obtain in this country clinical teachings in eye, ear, nose and throat diseases outside of two of our Eastern cities. The advantages from these clinics were curtailed by lack of material and equipment. Often in the early days if some of those who, at that time, were in a measure qualified in the work, were obsessed with the idea of the nonethical man (although medical ethics at that time were below par); that the man who could display the most glaring falsehoods in the daily press regarding his successful treatment of eye and ear diseases, was on the only road to success. Today, fortunately, the men qualifying for this work possess better ethical education, have a better academic foundation, and are more profoundly interested in the scientific professional success than in the financial.

At the time of the organization of this society, the West was

rapidly becoming more populous. With this movement there was a rush of young professional men, ambitious, scientific and well qualified to practice their specialty. Realizing the advantages accruing to them in a scientific and social way in belonging to a well organized society—being too remote from the place of meeting of one of the already existing societies and seeing the restrictions in membership, with almost insurmountable requirements in the other—made it obvious to many that the only remedy was the organization of a new ethical society, with requirements sufficient to restrict the membership to those in special work.

With the preceding as a fore-word, I shall proceed to interest you with a brief history of the Society for the first twenty years, with the growth and interesting data with which the Society during its life has been replete. To Dr. Hal Foster, of Kansas City, Mo., belongs the honor of taking the initiative in the organization of this Society. In January, 1896, Dr. Foster sent 100 letters to men in the West, Middle West and South, inviting them to meet in Kansas City, Mo., April 9, 1896, for the purpose of organizing a Society. These invitations were intended for only those who restricted their practice to diseases of the eye, ear, nose and throat. About thirty representative men of this section met at the Midland Hotel, in Kansas City, April 9, 1896, being nearly one-third of those invited. Many others wrote Dr. Foster enthusiastic letters, heartily endorsing the move. I consider it my good fortune to have accepted Dr. Foster's invitation and to have become one of the charter members. This, then, was the birth of this Academy, today the largest, best and most democratic special Society in the world. So instilled were the promoters of this new Society with Western ideas, that the first program announced the meeting of the Western Society of Eye, Ear, Nose and Throat Surgeons. The meeting was called to order by Dr. J. H. Thompson, president of the Kansas City Academy of Medicine. An Address of Welcome was delivered by Dr. C. Lester Hall, president of the Missouri State Medical Society. Dr. R. S. Black, president of the Kansas State Medical Society, responded on behalf of that Society. Adolf Alt was elected chairman by acclamation, Hal Foster, secretary. To Dr. C. Barck of St. Louis, belongs the honor of having read the first paper in this Society, his subject being, "Two Cases of the Opening of the Lateral Sinus for the Removal of Infectious Thrombosis; Recovery in One Case." The program for this first meeting was a long one, comprising many papers of great scientific value. There were twenty-nine

papers on the program, twenty-two of which were read by the authors, and two read by title.

At the business meeting April 10, the organization was named and officers elected. The question of a name for the Society occasioned a lively discussion. It was finally, by a large majority vote, christened the Western Ophthalmological, Otological, Laryngological and Rhinological Association. The weight of the name was enough to sink the Society into oblivion; mysterious to the laity, unpronounceable in a single breath, and too long. Its virtue was that the desires of the different "ologists" present were fulfilled. The Society, owing to its name, became popularly known and dubbed the "W O O L" Society. Its past record has fully demonstrated that it was up to the wool standard.

The first regular officers elected at this meeting for the ensuing year were Adolf Alt, St. Louis, president; W. C. Pipino, Des Moines, Ia., first vice president; B. E. Fryer, Kansas City, Mo., second vice president; J. H. Martindale, Minneapolis, third vice president; W. L. Dayton, Lincoln, Neb., treasurer; Hal Foster, Kansas City, Mo., secretary. St. Louis was selected for the next meeting place. It had been determined by vote that this Society meet in early spring, prior to the various State Medical and American Medical Association meetings. April 8-9, 1897, were designated for the second annual meeting. A Constitution and By-Laws were adopted as recommended by the Committee.

The second annual meeting was held at the Planters' Hotel in St. Louis. Dr. F. T. Lutz of St. Louis, in a few well chosen words, welcomed the members to St. Louis. A. C. Corr, president of the Illinois State Medical Society, responded on behalf of the Society. President Alt as his address, read a paper on "Hemorrhagic Glaucoma," and in closing recommended to the Society certain changes in the Constitution and By-Laws adopted at the first meeting. A committee consisting of Drs. Corr, Fryer and Dayton was appointed. They reported and recommended the adoption of the following:

ARTICLE 3.—"Amend words one year to three years; add, all applicants must be eligible for membership in the American Medical Association, and all applications for membership must be endorsed by at least three members of this Association who are in good standing."

This was the most important amendment of the several recommended inasmuch as it indicated that the very base of this

Society's foundation was loyalty to the A. M. A. The standard requirement for membership was raised from one to three years of special practice before being eligible for membership. This was an advancement and was the means of attracting many excellent young men to this Society, many of whom today are among the foremost workers in the Academy. At this second meeting seventy-five papers were on the official program. Only twenty-eight were read and discussed; this being a sufficient number to take up all the time of the Society. Many of the papers omitted were not by bona fide members, but by applicants for membership who had not paid their dues and had not been accepted by the Society.

It was apparent that many had sought a prominent place on this program for the advantage that might be gained through this medium of advertising. Prompt action on the part of the Society prevented a recurrence; this was done by suitable amendment to the By-Laws. The Society was called on to mourn the loss of one of its brightest and most vigorous workers, W. C. Pipino of Des Moines, Ia., the First Vice President, who was accidentally killed by being thrown from his horse on the streets of Des Moines during the preceding summer. The growth of the Society, while slow, had been progressive. At the second annual meeting twelve new members were accepted. B. E. Fryer, Kansas City, Mo., was elected president; J. Elliott Colburn, Chicago, first vice president; Frank M. Rumbold, St. Louis, second vice president; A. E. Bulson, Jr., Fort Wayne, Ind., third vice president W. L. Dayton, Lincoln, Neb., treasurer; Hal Foster, Kansas City, Mo., secretary.

The third annual meeting of the "W O O L" Association was held in Chicago, April 7-8, 1898—President B. E. Fryer in the chair. The meeting from a scientific viewpoint was a very successful one. The paper by Dr. Alt on "Research into the Histo-Pathology of Trachoma" was ably discussed by Gifford and Fryer. The paper of President Fryer entitled the "Antiseptic Preparation of the Conjunctiva for Cutting Operations on the Eye Ball" brought forth more discussion than any other paper. Dr. H. Knapp, who had already spoken briefly on this paper, by request gave the Society a more extended résumé of the use of antiseptics in the eye and in operations for cataract in particular. A resolution had been adopted at the second annual meeting accepting a proposition of M. A. Goldstein and Adolf Alt for the publishing of the transactions of the Society for that year. The contract was renewed for another year at this meeting.

Those of the faithful who were present in New Orleans at the fourth meeting will no doubt recall the chilly reception we were given by the weather man. With the thermometer hovering around 20 degrees below zero in the North when we started southward it was indeed a pleasure to anticipate the joys and comfort of a sojourn in the sunny South. We were in a measure destined to be disappointed, a "Northwester" had preceded us and the weather was extremely cold, the beautiful subtropical plants, trees and flowers, the pride of the city, were all frozen, ice covered the streets, business was at a standstill; no vehicles moving, no walking along the slippery ice covered sidewalks. Those who were fortunate enough to be quartered at the larger hotels were grouped around the radiators wishing themselves back home gathered around their own warm hearthstones and resenting the call of the Sunny South. The next day, the first of our meeting, the terrors of the "Northwester" had given way to the warm rays of Old Sol, the city awoke from its lethargy and things took on a brighter hue. This meeting was held during the Mardi Gras and each member was furnished invitations to all of the social functions given during these festivities. Those members who had the privilege of attending the reception given by Dr. Scheppergrel at his residence will never forget that pleasant evening spent amid the beauty and the chivalry of the South. This meeting was a great success both socially and scientifically. The meeting was called to order by Dr. Scheppergrel of New Orleans, first vice president. The President, J. E. Colburn, was unable to be present on account of illness. The Society had at this meeting as a guest of honor, George T. Stevens of New York, who addressed the Association on "Historical Notes Relating to Anomalies of the Eye Muscles," which elicited a very interesting discussion. There were thirteen new members admitted, and George T. Stevens of New York was made honorary member.

In our fifth year our Society was tottering; our finances were at the lowest ebb; dues were coming in slowly and expenses increasing. The necessary expenses for the meeting were paid by one of the officers who was compelled to depend on the collection of dues for his reimbursement. The outlook for the future of the Society was not a rosy one to contemplate. Former staunch members were becoming lukewarm in their support of the Society. The question arose whether to extend the territory from which we might invite members to join our Society or to continue as heretofore in the West and Middle West. Fortu-

nately it was proposed to extend an invitation to all reputable physicians in the United States, duly qualified in the practice of our specialties. This proposition met with almost unanimous approval. The next and most important point, perhaps, was the selection of a suitable and well qualified man for secretary. The selection made was most fortunate for the Society, the true hustling qualities of our late fellow, W. L. Ballenger of Chicago, being well known to you all. From the time of his election he began a propaganda to increase the membership of the Society. How well he accomplished his task is shown by the results; the two years he was secretary the Society gained 125 new members.

The fifth annual meeting was held at St. Louis at the Planter's Hotel, April 5-6-7, 1900. M. A. Goldstein, St. Louis, second vice president and chairman of the Committee of Arrangements, called the meeting to order and introduced Dr. Funkhouser, president of the St. Louis Medical Society, who welcomed the Society to St. Louis. President Scheppergrel then gave an address on the "Physiology of Vocal Production." A long list of new members were added at this meeting. The sudden death of Dr. Max Thorner being reported, the following resolution was adopted:

"We most respectfully submit for your consideration these tokens of respect and esteem tendered to the memory of our late able and estimable colleague and fellow member, Dr. Max Thorner of Cincinnati. In this bereavement our Society has lost one of its most valuable members and friends, the entire medical profession one of its most able champions and the laryngological world one of its brightest stars. Be it

Resolved, That this Society expresses its appreciation and esteem by spreading these resolutions upon the minutes, and that a copy of these resolutions be transmitted to the family of our dear friend. M. A. Goldstein, Edwin Pynchon, C. R. Holmes, Committee."

The officers selected for the next meeting were M. A. Goldstein, St. Louis, president; H. V. Würdemann, Milwaukee, first vice president; Fayette Ewing, St. Louis, second vice president; C. R. Holmes, Cincinnati, third vice president; W. L. Ballenger, Chicago, secretary; W. L. Dayton, Lincoln, Neb., treasurer.

The Society met in Cincinnati, April 10-11-12, 1901, and was welcomed by Dr. C. R. Holmes, chairman of the Local Committee, who introduced Dr. Dandridge, president of the Academy of Medicine, who formally welcomed the Society to Cincinnati. The address of President Goldstein was very interesting and contained many valuable suggestions looking toward the progress

of the Society. At this meeting steps were taken to drop from the membership of this Society after due notification of their delinquency, all members delinquent for two years in annual dues. The meeting of 1901 was the banner meeting as regards the admission of new members, there being forty new members added; the scientific program was replete with interesting papers and the attendance far above the average. The success of this meeting was largely due to the energetic work of Christian R. Holmes as chairman of the Committee of Arrangements. He put forth every effort to interest his friends and local colleagues in the Society and they responded nobly, with the result that a greater number of new members were admitted than at any other single meeting of the Society. The selection of officers for the ensuing year were C. R. Holmes, Cincinnati, president; W. L. Dayton, Lincoln, first vice president; J. L. Stillson, Indianapolis, second vice president; H. W. Loeb, St. Louis, third vice president; W. L. Ballenger, Chicago, secretary; O. J. Stein, Chicago, treasurer.

The Society met in Chicago, April 10-11-12, 1902, for its seventh annual meeting. Meeting was called to order by C. D. Wescott, Chicago, who introduced the president, Christian R. Holmes of Cincinnati. Dr. Holmes proceeded to congratulate the Society on the high character of its scientific work and on its rapid growth, stating that the number was increased by 40 members, who joined in Cincinnati, and that already at this meeting 75 applications were in. I take the liberty of quoting a paragraph from Dr. Holmes' speech, because it so closely conforms to my ideas:

"That there is a need for an association of this kind is beyond dispute. There are a great many men throughout the West who seldom or never go East. And besides, in the American Ophthalmological Association, a limited number only are admitted. It will redound to the good of the profession throughout the West if we have a good scientific association, admitting to its membership any physician who has been duly qualified, and who has conducted himself according to the ethics and complied with the requirements of the A. M. A. Active membership in an association of this kind improves the man and the profession at large, as no man can participate in the deliberations of a society like this without benefit to himself and his patients."

The growth of this Society has been due to the energetic work of its officers. Dr. Holmes was ably supported by Secretary Ballenger and Treasurer Otto J. Stein in his arduous campaign for new members. Dr. Holmes instead of giving the

regular address gave an interesting stereopticon lecture on the "Development of the Ear from the Lowest Form of Animal Life up to and including Man."

New wants and emergencies having arisen owing to the rapid growth of the Society, a committee on Revision of Constitution and By-Laws was appointed, consisting of Casey A. Wood, Adolf Alt, J. O. Stillson, B. F. Fryer, Edward Jackson, Harold Gifford, H. V. Würdemann, this committee to report at the next annual meeting. The *Journal of Ophthalmology* and *The Laryngoscope* were continued the official organs of the Society. The officers elected were W. L. Ballenger, president; J. O. Stillson, first vice president; J. M. Ray, second vice president; Edwin Pynchon, third vice president; Derrick T. Vail, secretary; Otto J. Stein, treasurer. Dr. Ballenger in assuming the duties of President, carried with him from the secretary's office that he had so ably filled the past few years, a complete knowledge of the Society's affairs. His wide acquaintance with all those engaged in the practice of oto-laryngology was invaluable and proved of great benefit to the Society.

It was quite proper that Dr. Ballenger, being a native Hoosier, should hold this position of honor and trust in a city on his native heath and Indianapolis, Ind., April 9, 1903, found the "W O O L" Association meeting with them for the first and last time, for an important event in the history of the Society was portending. The committee appointed at the last meeting for a revision of the Constitution made their report, which after a few minor changes was adopted, and the American Academy of Ophthalmology and Oto-Laryngology was brought into existence. The Constitution adopted at this time is the one which we are working under today, with the addition of several amendments. Fifty new fellows and members were admitted at this meeting. The officers elected for the ensuing year were Edward Jackson, president, Denver, Colo.; Dudley Reynolds, first vice president; J. J. Kyle, second vice president; John W. Murphy, third vice president; Derrick T. Vail, secretary; Otto J. Stein, treasurer. Members of the first Council were Adolf Alt, W. L. Ballenger, Casey A. Wood, Christian R. Holmes. Edward Jackson not being present at the meeting was telegraphed of his election; as unsought honor was thus thrust on a most deserving member of this Academy. Dr. Jackson in a telegram accepted the presidency, thanking the Society for the honor conferred. The meeting was placed at Denver and held in August in order to give the members the benefit of a visit to this beautiful mile-

high city during the heat of the summer and to provide an outing to those who desired, in the mountain resorts near by. All sections of the country responded and many of our eastern confreres viewed the snow-capped summit of the famous Pike's Peak for the first time. This session of the Society was one of the very best ever held from the standpoint of scientific value, social enjoyment and attendance. The meeting convened Aug. 24, 1904 for a three day session with Dr. Jackson in the chair. Eighty-two members were admitted and by a special provision in the Constitution the following were declared Fellows by the Council, and elected by unanimous vote: A. A. Hubbell, Emil Mayer, R. A. Randall, C. W. Richardson, George E. de Schweinitz. A committee composed of Drs. Hotz, Barken and Weeks were appointed to extend a special invitation to the International Ophthalmic Society to hold its next meeting in this country; the Academy joining with the American Ophthalmologic section of the A. M. A. in the invitation. A committee consisting of Drs. Lockhard, Loeb and Shurley was appointed to represent this Academy at the celebration of the fiftieth anniversary of the invention of the ophthalmoscope and the 100th birthday of Garcia. H. W. Loeb was selected for president for the ensuing year; D. T. Vail, first vice president; Robert Levy, second vice president; Eugene Smith, third vice president; George F. Suker, secretary; Otto J. Stein, treasurer; Council, Drs. Jackson, Balenger, Good and Ray.

Eastward on Eastward was evidently the slogan of the Academy, gathering the very best from all sections of the country under its banner of democracy. The large attendance at Buffalo at the tenth annual meeting, Sept. 14-15-16, 1905, with the admission of ninety-three new members, clearly demonstrated that our eastern colleagues were becoming interested in the Academy. The Academy met for the first time in two sections—President Loeb as chairman of the ear, nose and throat section, with J. J. Kyle, his secretary; Derrick T. Vail, first vice-president, as chairman of the eye section, with George F. Suker, his secretary. Dr. Herman Knapp delivered a paper on the "Lens Capsule in the Operation for Cataract," which brought forth a long discussion. Dr. G. Lindsay Johnson of London, England, was recommended and elected an honorary member. Drs. Alt and Goldstein were continued as publication committee with *The Laryngoscope* and *Journal of Ophthalmology* as the official organs.

The eleventh annual meeting was held Aug. 30, 31, and Sept.

1, 1906. This meeting had been accredited to Detroit but the Committee of Arrangements with Eugene Smith, vice president, decided that St. Clair, a near-by pleasure resort on the St. Clair river, offered many advantages; hence he located the meeting at Hotel Oakland at St. Clair. The meeting was welcomed in an exceedingly pleasant manner by Congressman Edwin Denby of that district with a response by C. P. Stockwell, president of the Michigan State Medical Society. An important amendment to the constitution was adopted at this meeting. It had been evident to some members of the Academy that politics were entering into the management of Society affairs to such an extent that the democratic principles that had heretofore prevailed were threatened with destruction; an amendment was therefore introduced to better provide against such a calamity. Believing that more representation in the Council from the Fellows on the floor would be beneficial in the conduct of the affairs of the Council, representing more truly the body politic of the Society, the following amendment was adopted:

"That there shall be a Council composed of the President, who shall be Chairman; two ex-presidents next preceding him, and four Fellows of the Academy, two to be chosen each year, and to serve for a term of two years."

With the adoption of this amendment the balance of power lay in the Fellows from the floor, rather than in the ex-presidents—truly a boost for democracy.

Dr. Emil Mayer, of New York, was appointed to represent this Academy at the celebration of Frankel's birthday. He was given power to formulate an address, to be signed by himself and the president and secretary. A committee was appointed to assist in carrying out the suggestions of F. Park Lewis in the prevention of ophthalmia neonatorum, Dr. Lewis to be chairman to work in conjunction with a like committee from the American Medical Association. Mr. Marcus Gunn of London, England, was the guest of honor, and gave us a very interesting address on "Optic Nerve Ailments." Dr. Dundas Grant of London, Eng., was also present at this meeting by invitation and addressed the meeting on "Some Practical Problems of Otology and Rhinology." Messrs. Gunn and Grant were our first foreign guests by invitation and were made honorary members. A proposition of H. V. Würdemann to print the Academy's proceedings was accepted. The following officers were elected for the ensuing year: J. A.

Stucky, president; George F. Suker, secretary; Otto J. Stein, treasurer.

At the urgent request of our Kentucky contingent, Louisville was selected for the twelfth annual meeting and on September 26 the Academy convened in that city with J. A. Stucky of Lexington, president; George F. Suker, secretary. The program was replete with exceedingly interesting papers and the social features were all that could be desired.

A very interesting symposium on otitic diseases was given at the twelfth meeting of the Academy. President Stucky presented "Some Clinical Observations in Intracranial Complications of Otitic Origin"; James F. McKernon, "Pyogenic Diseases of the Brain of Otitic Origin"; "The Ocular Symptoms Following Intracranial Complications of Otitic Origin," was presented by Dr. Percy Friedenbergl; Dr. Sprague, of Lexington, Ky., discussed "Diseases of Otitic Origin from a Neurological Standpoint." At the opening of this meeting in Louisville none of the three vice presidents was present, and our genial friend Dr. Savage, of Nashville, was called on to act as chairman of the eye section.

Thirteen had no terrors for Derrick T. Vail when he called the thirteenth session of the Academy to order at Cleveland, Aug. 27, 1908. Dr. Vail had served the Society well as secretary and had brought with him to the president's office the hustling qualities that made him a successful secretary. At the opening of the session Dr. Vail stated that it had been decided that it was not necessary to have an Address of Welcome, the welcome being rather expressed by the work of the local committee in the splendid arrangements made by it. It was resolved at this meeting that the interest or profit which accrues on the surplus funds of the Society be at the disposal of the Council for the purpose of defraying the expenses of original investigations deemed by the Council as worthy.

"Resolved, That the expenses of the entertainment of this Society in any city in which they may meet be paid from the funds of the Society rather than by the local committee."

Mr. J. B. Lawford of London, Eng., our guest, delivered a very interesting and scientific address on "The Etiology of Choroiditis." Mr. Lawford was unanimously by a standing vote made an honorary member. Mr. Lawford in accepting the honor said:

"I am very pleased and very proud to become an honorary member of the Academy and I trust I shall have the honor of visiting you again."

The officers elected for the coming year were Otto J. Stein, president; Percy Friedenbergr, first vice president; D. W. Green and C. Barck, second and third vice presidents; George F. Suker, secretary; Secor H. Large, treasurer.

The election of Otto J. Stein was very gratifying to all of the members of the Academy. His efficiency as treasurer of the Society for the past seven years was assurance enough to all that his election to the presidency was a fitting recognition of a faithful and efficient officer.

The fourteenth annual meeting was called to order by President Otto J. Stein, Oct. 4, 1909, in New York City, at Hotel Astor. Dr. McKernon of New York presented to the Academy Dr. John A. Wyeth, president of the New York Academy of Medicine, who welcomed the Academy to New York. The secretary in his report congratulated the Academy on the *Transactions* which were published by the Academy, the work being done by the A. M. A. printing office. The publication by the Academy of its *Transactions* was a move in the right direction and has been followed up to the present time, giving general satisfaction. The secretary also reported that our total membership was now 600.

The president in his address suggested some changes in the Constitution and a committee of five was appointed to consider these resolutions. F. Park Lewis of Buffalo introduced a resolution that a committee be appointed representing this Society, to cooperate with popular and medical bodies for the purpose of securing information and devising measures for the prevention of avoidable accidents which may result in blindness. Dr. Juan Santos Fernandez, Havana, Cuba, was a guest of the Society and addressed them on "Some Observations in Thirty-five Years of Service in Cuba Pertaining to Ophthalmology." Professor Onodi, Budapest, and Dr. John Sendziak, Warsaw, Poland, were our other invited guests, who, however, had been prevented by illness from being present. The address of Dr. Sendziak on the "Progress of Laryngology and Rhinology Since the Invention of the Laryngoscope, with Special References to the Participation of America in this Progress," appeared in the annual proceedings of the Academy for 1909. Dr. Fernandez was made an honorary member of the Society.

Dr. Percy Friedenbergr presented the Society with a special de-

sign for an official badge that was both original and distinctive. The badge was adopted as the official one and has been in use since that time. On recommendation of the Council the Academy voted an honorarium to the secretary for the past year. The Constitution was so amended at the next annual meeting that the secretary received an honorarium yearly.

For the second time in the history of the Society the meeting was held in Cincinnati, Sept. 19-20-21, 1910, when President Wendell Reber called the fifteenth session of the Academy to order. The president and Secretary Suker determined to make this meeting a record breaker in every way and as the result of their enthusiasm for the welfare of this Society they presented a scientific program of great worth to all the Fellows of the Society.

We were honored by having Mr. Sidney Stephenson, London, Eng., as our guest, who delivered the anniversary address "On Sloughing Cornea in Infants, an Account Based upon the Records of Thirty-one Cases." Dr. Stephenson was made an honorary member and presented with the first gold medal ever given by this Society, inaugurating a custom afterwards followed with all of our invited guests. President Reber in his address made several recommendations, one of which provided for the establishment of a fund to be awarded either annually or bi-annually to any member of the Academy who shall have made some signal contribution to ophthalmology or oto-laryngology. This award was intended to encourage the younger members to original research with the idea of rewarding such research in some suitable manner.

The sixteenth annual meeting was held Sept. 26-27-28, 1911, in Indianapolis, Ind., J. J. Kyle, president, and George F. Suker, secretary. Elaborate arrangements had been made for the entertainment of the Society by the Committee of Arrangements so that besides having an intellectual feast all of the members and their ladies received every attention socially. We had as our guest of honor Dr. Albert Gray of Glasgow, who delivered the anniversary address entitled "Problems of Oto-Sclerosis and Allied Conditions." Dr. Gray entered deeply into the subject and by aid of his charts on heredity showed conclusively the process and progress down the line of ancestral heredity. His address was particularly well received by the otologists present. The report of M. A. Goldstein of St. Louis, who was a delegate to the general Otological Congress, was read. From the fact that the State of Indiana had enacted a law looking toward the

prevention of blindness, a resolution by Dr. Wendell Reber was presented and adopted:

"Resolved, That we heartily congratulate the people of the State of Indiana that the Legislature of 1911 gave them such a wise and efficient law intended to prevent blindness."

Drs. Kyle, Stein, Kopetsky, Ballenger and Hitz were appointed delegates to the International Otological Congress, meeting at Boston in 1912. The sad news of an accident to Dr. Gibson and wife while en route to this meeting in an automobile, was announced to the Society. A message of sympathy was ordered sent to the family.

The first meeting held outside the boundaries of the United States was held at Niagara Falls, Ontario, Aug. 20-21-22, 1912. Dr. George F. Suker, after eight years of arduous labor as secretary—a labor of love for Dr. Suker—was honored by the Academy by being chosen as its president for this year. In his speech of acceptance Dr. Suker declared that he wanted to live to see the membership of this Society raised to 1,000. His desire has been fulfilled and we are proud to note that Major Suker is still with us and in the service of his country. Dr. Anton Elschnig of Prague, Bohemia, was our invited guest and gave the anniversary address on "Sympathetic Ophthalmia." Mr. Heath of London, Eng., was also present and was invited to participate in the program and at the request of the president entertained the Academy by elucidating further his work on the mastoid. Dr. Elschnig was presented with a medal and made an honorary member. The Academy had jealously guarded its honorary roll and but two Americans had thus far received this signal honor, Herman Knapp and George T. Stevens of New York being those Americans. The Council having recommended for honorary membership a man who has the love and respect of all of his confrères in our broad land; because of his achievements in his profession and his wonderful personality, the Academy conferred on Dr. Samuel D. Risley by a unanimous standing vote, an honorary membership. The Council recommended that \$2 of the annual dues of each member be devoted to the *Monthly Index and Abstract of All the Literature in Eye, Ear, Nose and Throat*; a copy of which is to be sent to each member whose dues are paid by February 1 of the ensuing year. The recommendation was adopted by the Academy. Edwin Pynchon of Chicago and C. E. Evans of Union City, Ind., were made life members, this being the first instance in which the

Council used the power given them in the Constitution. At this meeting Risley, Jackson and Lewis were appointed a committee to formulate a definition of blindness. For the first time in the history of the Academy the nomination for the new officers as recommended by the Council was not accepted by the body politic, who took the power delegated them by the Constitution and nominated and elected a president from the floor. The Academy made no mistake in electing J. W. Murphy of Cincinnati to the chair. The manner in which he was selected was sufficient evidence of the high esteem in which he was held by his Fellows in the Academy.

The Academy convened at Chattanooga, Tenn., Oct. 27, 1913, for a three day session with J. W. Murphy, President, and Lee Masten Francis, Secretary.

We had as our guest of honor Lieutenant-Colonel Elliott, late of Madras, India, who gave the anniversary address on his "Methods of Trephining in Glaucoma." He requested a thorough discussion and the same was entered into by a large number of the members present. Colonel Elliott was then presented with the Academy medal and made an honorary member. An important amendment to the Constitution was offered:

"Resolved, That a member of the Council shall not be eligible to the nomination for President during his term as Councilman."

Our meeting in Chattanooga was particularly interesting from the scientific point of view. Wendell Reber, ever eager to advance the cause of medical training in our colleges and post-graduate schools, introduced the following:

Resolved, That the American Academy of Ophthalmology and Oto-Laryngology realizing that standardization of Post-Graduate Medical teaching in Ophthalmology and Oto-Laryngology is much needed, feels that definite action in this direction should be taken. *Resolved, further*, That it is the sense of this meeting that two committees be appointed by the chair to confer with similarly appointed committees from the American Ophthalmological Society and the sections of Ophthalmology and Oto-Laryngology of the A. M. A. on the one hand, and similar committees from the Oto-Laryngological Societies. The end in view will be to induce the various Post-Graduate Institutions of the United States to adopt some manner of uniform curriculum and uniform requirements for admission to Ophthalmic and Oto-Laryngologic practice, said committee to report back to the Council at the Boston meeting.

The committees appointed were Drs. Reber, Jackson and Lancaster for the ophthalmologic, and Drs. Ingersoll and Bal-

lenger for the oto-laryngologic section. A pleasant feature of this meeting was the visit of the members and their ladies to Look-out Mountain and Missionary Ridge battlefields, the trip being arranged so that it would not break into the session of scientific work.

The nineteenth meeting of the Society convened at the Hotel Copley-Plaza, Boston, Oct. 19, 1914, for a three days session, Dr. J. M. Ray, president. C. J. Blake welcomed the Academy to Boston. Dr. Blake gave in detail a history of the advancement in medicine made in Boston, and the advantages in medical training owing to the establishment of many excellent hospitals.

The excellent work done by Dr. Greenwood and his Committee in providing many instructive and interesting clinics in the various hospitals was fully appreciated by the Academy. Wendell Reber, chairman of the Committee on Post-Graduate Teaching and Ophthalmic Examinations, made his report. Dr. Reber said in closing:

"The Committee feels that the function of the Board should be to examine and then certify, and not to confer degrees. We all have our degrees, of which we are proud."

The report was adopted and the following were appointed as the first Board of Examiners:

Ophthalmic: Wendell Reber, three years; Walter B. Lancaster, two years; Frank C. Todd, one year.

Oto-Laryngologic: J. M. Ingersoll, three years; Samuel Iglauer, two years; B. R. Shurley, one year.

A letter from Colonel Elliott of London, Eng., was received by President Ray and read before the Academy. Colonel Elliott sent his best wishes for the success of the meeting and expressed his pleasure and pride of being an honorary member of the Academy.

Report was received of the death of Edwin Pynchon, a pioneer in this Society, a man highly esteemed by his fellows.

The twentieth meeting of the Academy at Chicago, Oct. 5-6-7, 1915, was called to order by President Joseph Beck. He congratulated the Academy for opening on time and declared that there was no need of an address of welcome, that the entire medical profession and the members of the Academy in Chicago welcomed us and invited us to enjoy the hospitality of the city, emphasizing the fact that our official badge would be an open sesame to everything within the municipal boundaries. Thursday was given over to clinics in the different hospitals.

The most important matter to come before the Society was the report of the Committee on Education. The report was adopted and a Board of Examiners appointed as already mentioned. An amendment to the Constitution and By-Laws making the treasurer an ex-officio member of the Council was adopted at this meeting. Death had levied a heavy toll on our colleagues the past year. C. P. Johnson, Coffeyville, Kan.; Charles A. E. LeSage, Dixon, Ill.; Fred A. Guthrie, LaSalle, Ill.; Mark E. Stevenson, Akron, O.; Benjamin Gleason, Danville, Ill.; Sherman Vorhees, Elmira, N. Y.; Dudley S. Reynolds, Louisville, Ky., had passed to the great beyond during the year 1914.

It is not fitting that we should close this historical résumé of twenty years of this Academy without speaking in memoriam of Dr. Wendell Reber, that grand man who practically sacrificed his life to this Society; a man who by his own energies, by his wonderful powers of assimilation, his clear and concise reasoning, his capacity for study coupled with an indomitable will, rose from an obscure country physician to one of the foremost ophthalmologists in this country. He was present last year at the Memphis meeting and apparently in the best of health; he remained after the session to assist the Board of Examiners, of which he was chairman, to hold the ophthalmological examinations. He contracted a slight cold in Memphis which was increased by his trip home and soon after his arrival home he was compelled to take to his bed, stricken with pneumonia.

Dec. 30, 1916, Wendell Reber passed on. His cheery smile and his ringing voice never to be heard again in the halls of this Academy. "To live in the hearts we leave behind us is not to die."

Your Council at its meeting in New York City, June 8, 1916, imbued with the spirit of democracy, the keystone of this Society, desirous of putting this Society on record as ready to do its bit in this world war, offered to present to our government a fully equipped base head hospital. The Surgeon-General decided that the government would establish a base head hospital in place of accepting one from the Society. He declined the proffer of the Academy and thanked the Council for their patriotic offering. The Council may make some further recommendations to the Academy along these lines at this meeting.

Many of our confrères have responded to the call of duty and have entered into the service of their country, leaving behind them wives, sweethearts and business, regardless of the future, with no thought of tomorrow save one, our country's need. To

those who thus represent us in our country's service I recommend that the Academy empower the Council to remit all dues during the period of the war. I believe that it would be appropriate at this time to suggest that the Academy hold its twenty-fifth meeting in its birthplace and I recommend, therefore, that this matter be taken up seriously by the Academy and if all conditions are favorable that we arrange, at the proper time, to hold our twenty-fifth meeting in 1920 at Kansas City, Mo.

Since the organization of this Society in 1896 we have lost by death three ex-presidents, B. E. Fryer, Wm. L. Ballenger, and Wendell Reber.

“The Moving Finger writes;
And having writ, Moves on;
Nor all your piety nor wit
Shall lure it back to cancel half a line
Nor all your tears wash out a word of it.”

EXPERIENCES IN FRANCE*

MAJOR ALLEN GREENWOOD, M.R.C., U. S. ARMY.

Your president, in introducing me, made one mistake. This is not an entertainment. It seems almost a crime to step in here after your pleasures of the evening, to tell you a little bit of what this war actually means. I do not believe the average citizen of the United States knows what is going on, or what may come. Especially in view of the happenings of the past few days. It seems a pity to spoil your dancing by coming in with the story I am going to tell you. But I have been in London in 1915 and 1916, where nobody is dancing and nobody is expected to dance.

I stayed a few days with Lieut. Col. Elliott, who is carrying on his work as though nothing had happened, but he has lost seven of the male members of his immediate family. I also stayed several days in one of the small hotels in London where lives a member of the House of Lords. I talked with him and did not know that a few hours before he had received word that his last remaining heir had died at the battle of Jutland. That is what the war is meaning to Great Britain. I hope the Divine Power will not ask us to go through such things for long. War is a serious business, and this is the most serious war this world has ever known, and we are only beginning to realize in this country what it means.

It is the realization of this that has led many of your members as well as other medical men to enter the service, so that at the present moment there are on the rolls of the Surgeon General's office fully fifteen thousand medical men who have enlisted to take care of the wounded. An order has recently gone out that no more commissions will be issued for the present, until things are straightened out, and better arrangements are made for the men already commissioned and on active duty.

Another thing that will interest you is that courses of instruction are to be established, and the men in the base hospitals and in the regimental units will attend these courses or postgraduate schools at the base hospitals, where they will receive instructions in all the branches of medicine.

*At ten o'clock, at the close of the dinner-dance, Dr. Dayton called the guests to order, introducing Major Greenwood, who had been delegated to attend this meeting by the Surgeon General of the United States Army.

The subcommittees on ophthalmology and oto-laryngology have drawn up a syllabus for the courses to be given medical officers in ophthalmology and otology. This country is being equipped in a way that will make it possible to send to France men highly specialized in every line. Intensive classes in training in brain and oral and plastic surgery are also being conducted in several of the large medical centers of the United States for men of the Medical Reserve Corps.

Men who enter the Medical Reserve Corps, or who contemplate it, must bear in mind that work in the war zone is vastly different from that in general practice. All are medical graduates with an education supposed to fit them to meet the medical and surgical emergencies that arise. It is wholly needless to point out the inestimable value of having them do the special work they are fitted to do along special lines. But in this time of stress the medical man must look upon his preferences as entirely subordinate to the general good of the service. The internist may find his experience in orthopedics gained years ago of great value for he may find himself working along this line. The genito-urinary surgeon may develop into a brain surgeon, and the ophthalmologist may put his knowledge of sanitation to useful account or he may assist as an anesthetist. First in importance is the preservation, as far as possible, of life and the prevention of unnecessary crippling, and secondly, the men must be as quickly as possible restored to the fighting force, or as many of them as possible. Among the big restoration hospitals where the work is carried on by highly trained experts, there should be a wonderful *esprit de corps*. The two summers that I spent in the field gave me an insight into the kind of work the specialist may run up against and be obliged to do.

There are spread along the coast of France about one hundred and fifty British Base Hospitals, equipped to take care of over a thousand men apiece. The hospital I had the fortune to serve in, which went over in the early spring of 1915, had a thousand beds. The following summer this was increased to twenty-three hundred beds, and I have seen them nearly all filled with men horribly torn, crushed, or burned by liquid fire. I can't draw for you a pen picture of what one sees after a big drive in France. No pen can picture it. There are wounded men coming in by the hundreds and thousands, and last Fourth of July there were lying in the field hospitals and clearing stations of France sixty thousand wounded men waiting to have Base Hospital attention, with the

hundred and fifty thousand beds full, so that many had to be sent direct to England. Hospital train after train was coming in, and a hospital of a thousand beds would have to treble in size and every surgeon and nurse have to put their energies to the utmost of human endurance. The ophthalmological surgeon had to help with the ether and anything else that came up. As Dr. Derby said in a recent letter, he had turned his hand to minor surgery.

I want to show you a few pictures, some not wholly pleasant, and I trust you will forgive me for showing you exactly what this surgical work means in France today.

(Followed a large number of lantern slides of wound operations and hospital scenes, all explained by Major Greenwood.)

I have shown you a little of what the war means. And yet, let me tell you what medical and surgical science can do in this war. Here was a hospital of twenty-three hundred beds, and in the summer of 1917 there passed through it ten thousand men, with a death rate of less than 1%. You all want to know about the eye cases. About 10% have some injury about the face that involves the eye. I have seen many men with both optic nerves cut by bullets, and have sometimes had to take out the remains of both eyes in the same patient. That was the saddest part to me. And yet we can by our conservative methods, and by getting at these men early, save a great many apparently hopelessly injured eyes. By the use of the giant magnet we can also save a great many of the eyes seriously injured by foreign bodies which the X-Ray localizes or the magnet demonstrates. And when you see one eye put out you feel how essential it is to do all you can possible to save a bit of sight in the remaining eye.

War is not a pleasant subject. I think I have kept you from your dancing a sufficient length of time, but will be glad to answer any questions I can. If any of you go to France you will feel the intense interest in the work, and any one of you will do what we have done—will put your shoulders to the wheel of Medical progress. And if some officer comes along, your junior in years and rank, perhaps one you have trained, you will willingly turn to as I have repeatedly done and etherize for him while he does a major operation. Every man must do just the very best he can for the good of the service and the cause of Democracy. It is so wonderful and so vital to try and see how many of the injured men you can put on their feet, or restore their sight, or hearing, to be sent home to be worth while, or to be able to do their extra bit for their country and the Cause.

Shell shock will send many in without hearing, sight or speech

and by giving them rest and suggestion they come out all right. Many come in with a slight injury to the skull and in a few days develop choked disc and convulsions, and a trephining has to be done. I had in my ward at one time in adjoining beds three men who had received the usual furrow wound of the scalp across the back of the head, made by a high velocity bullet. They all had a homonymous hemianopsia and optic neuritis. After I had done a trephining and removed the splintered inner table of the bone and the disorganized brain substance, they promptly recovered with some improvement in the hemianopsia. Meningitis rarely follows brain surgery where the nasal space or ethmoid cells or pharynx have not been opened. I have seen a case of gas infection of the brain get well without resulting meningitis due to the walling off of the dura. We have to get the shrapnel out of the brain, of course, if possible. Cushing is doing excellent work in extracting shrapnel from the brain by using the Lancaster magnet. Gas bacillus infection of wounds is usually the worst complication and comes when the outer wound is healing, and the tissues underneath become filled with gas and pus. I had five patients last summer who had gas infection of the orbit, and four recovered. I believe this country has solved this problem, however, with the antitoxin for the gas bacillus infection discovered at the Rockefeller Institute. This has nothing to do with the poison gas but is an infectious material taken from the highly cultivated soil of France, and is introduced on the dirt and cloth that is carried into the wound.

Another interesting observation was that we saw only one or two cases of tetanus in the ten thousand as a result of the universal use of antitetanic serum in the clearing stations.

I did not see a single case of sympathetic ophthalmia. If the vision was lost and the eye irritable, it was removed. Also, we found it better, if a man developed later an iridocyclitis, to remove all sightless eyes. This may account for the lack of sympathetic ophthalmia. I removed, during the two summers, nearly one hundred eyes, and never did a man object to having the operation performed. Not a single man has ever objected in my presence to any operation advised for him.

In the winter time in each one of the hospital tents we put small stoves, with a big sheet or iron replacing one side section of the tent wall and the pipe going through that. We only had one fire in the hospital, and that was due to one of the soldiers lighting a cigarette, so we had to stop their smoking in the ward. On sunny days we opened the side of the tent and shoved the men out into the sunlight.

ADDRESS OF WELCOME

EDWARD STIEREN, M.D.

PITTSBURGH

Fellows of the Academy: Our city has been called the workshop of the world; here many of the death-dealing instruments of war are being made. With a daily pay-roll of \$2,000,000 a considerable proportion of the financing of the war has come from Pittsburgh and we have given liberally of our men of science. Ere another year rolls around no doubt many of us assembled here today will be directing our peculiar skill to the relief of pain and suffering, to the reconstruction and rehabilitation of legions of distressingly mutilated fellow beings, to the end that democracy may be saved to the world.

Whether we are called beyond the seas, to cantonments or base hospitals in this country, or following our prosaic, but none the less important, duties among the industries at home we will be fulfilling our destinies and doing our bit to the best of our ability.

Hence it is that the government has sent messages encouraging the meeting of scientific bodies, such as this, that each may become more and more proficient in the work he has to do.

Mr. Wilson has said that he does not want this country to go into deep mourning just because we are at war and, while there will be little time or opportunity for frivolity, a little levity has been provided and will be indulged in after the day's session is over.

That you chose Pittsburgh for the meeting place this year and your humble servant to be your first vice-president is much to be proud of and I do assure you that I am deeply conscious of the double honor.

And yet a feeling of sadness, paramount to all other emotions of which I am conscious, possesses me, for Wendell Reber's absence will be a void felt by all.

It is a pity he was not spared to meet with us for many more years. His thoroughness and enthusiasm will be sadly missed, but this year especially, for we are meeting in his adopted State and it seems only a few months past when at Memphis he seconded the desire that this year's meeting be held here.

Were I required to epitomize his life I could do no better

than to quote from the concluding paragraph of his address as President of this society in 1910:

“Let us be thorough in all we do. Let us be strong and the exercise of our strength today will better equip us for tomorrow. Today is our day—your day and mine—the one day we have—the day in which to play our part. What our part may signify in the great whole we may not understand—but we are here to play it and now is the time.”

Wendell Reber's influence will be felt for many years to come and the Academy will thrive and advance because of it.

We are meeting this year under unusual conditions. We are meeting in the heat of the most gigantic cataclysm this old world has ever witnessed, and while many of our members are absent serving the Flag, nevertheless a program of unusual merit is offered.

That we are here in such excellent number typifies the great American spirit that, no matter what happens, necessary affairs must be prosecuted as usual.

I trust your visit to Pittsburgh will be remembered as not only profitable but pleasant and entertaining as well.

MALINGERING FROM THE STANDPOINT OF THE EYE AND THE EAR

ITS DETECTION. METHODS USED. A REVIEW

GEORGE F. KEIPER, A.M., M.D., F.A.C.S.
LAFAYETTE, IND.

The recent draft for soldiers to fill the ranks of the new National Army has revealed the necessity for a review of the tests used to detect malingering, at least so far as the eye and ear are concerned. The examination of over 1,200 men committed to our care, who were called for examination as to their qualifications physically, for military service, revealed numerous attempts made to defeat the purposes of the selective draft by malingering. The profession of medicine can ill afford to be deceived by the liars that they are to say nothing of their cowardice. Therefore, it is incumbent on us to thoroughly acquaint ourselves with all the phases of this subject that we may do our whole duty to our government, for many more men will be called to the colors and the boards of conscription will have further need of our services.

It has been my own experience that fully 90 per cent. of the men examined are thoroughly honest and respond promptly and truthfully to the tests imposed. Their patriotism is thus unquestioned.

The malingerer may appear before a physician prior to the date set for his official examination to procure an affidavit alleging disability sufficient to disqualify him from service, which he will take to the board of conscription. I may state right here that any doctor is very foolish to give such a paper for no standing will be accorded it there and if the board finds its allegations are untrue the doctor giving the certificate is placed immediately under suspicion of having given a biased certificate and for the fee involved, and in these strenuous times criticism is more rife than in times of peace.

It has been alleged that physicians have coached certain men in the art of malingering. It has been further alleged that certain members of our profession have instilled drops in the eyes of men producing cyclopegia and thus reducing vision below the standards required. Both frauds are so easily detectable that

any physicians guilty of such practices should be cited before their respective boards of medical examination and registration for revocation of their licenses to practice medicine.

It may be noted in passing that at times the possessor of real defects in vision and hearing may malingering in order to enter the service. We are informed that one young man in our state while waiting his turn for examination memorized the letters of the exposed test card, and when examined repeated from memory the letters when his blind eye was exposed. His zeal is to be commended though his methods are to be condemned, for such an one is dangerous both to himself and his future comrades in arms. Hence, it is best to have a room separate and apart from the room in which the general examinations are being conducted, in which to examine the eyes and ears.

Then, there is the type of the malingerer who fails to lie enough. He claims his sight and hearing to be poor but we are sure that he is lying from his general attitude. However, as he manifests enough vision and hearing to qualify him for service we pass him in and he wonders how it all happened.

Malingering is not confined to attempts to escape army and navy service, for persons malingering to claim damages not their due, or to procure pensions unwarranted, or children to escape school attendance, and the latter can be cured by making preparations usual to an operation.

We may now start with this general proposition: The malingerer is a liar and as such possesses all the limitations of the average liar. His feigned disability, as a rule, is easily detected. It may take some time, however, in a few instances.

The general principle involved is to make the examinee see and hear against his or her own will.

The tests fall under two heads: (a) Objective: namely, what the examiner sees and hears. (b) Subjective: that is, what the examinee sees and hears.

As these examinations are conducted by general practitioners in localities where the services of competent oculists are not available, we are compelled to deal with much elementary matter. In conducting such examinations we must first understand the requirements necessary for military service: the recruit must have at least 20/40 vision in one eye and in the other eye 20/100 vision without glasses or 20/100 in each eye if correctable with glasses to 20/40.

His hearing must be 10/20 of normal.

EXAMINATION OF THE EYES

The conscript is seated in the fashion with us 20 feet from the Snellen test card. One eye is covered with a card, not the hand. He is asked to read the letters in order from the top as far down as he can, and the result procured is recorded on the blank in the form of a fraction. While 90 per cent. respond readily, it is interesting to note the hesitancy of the malingerer, and how easy it is to make him read further down the card than he thinks he can. I use a card which at the side has the lines numbered with figures of the size of the 50 foot line. He may begin to read the 200 foot line and repeat the figure or ask if he shall repeat the figures. That already determines his vision to be 20/50. He may stop at the 50 foot line, but urging him on because he saw the other figures so well we thus persuade him to read the next line, which as it were pushes him over into the service, unconsciously, or if he discovers his error it is too late. Usually he does not discover the error committed. Then the other eye is similarly tested.

If in spite of these ruses the vision remains below the above standards, we must test each eye at 10 feet first, and then at 40 feet. If the conscript has a real defect the fractions obtained will be constant as a rule. Furthermore, by using the illiterate test type with a mirror so placed that when he is turned around to read the type in the mirror the apparent distance is trebled we have another method of detection for his ability to judge the apparent distance is now lost, for the type in the mirror seems closer than in reality, and he reads further down than he should if he were truthful. In the discussion of this subject before the recent meeting of the section of ophthalmology and oto-laryngology of the Indiana State Medical Association, the case of a malingerer was cited, to catch whom it was necessary to use one of the test cabinets exposing a single line of test type at a time.

If the vision is below standard we must examine the conjunctiva for trachoma, the lids for ptosis, blepharitis, ectropion, entropion, and the lachrymal apparatus for epiphora; the eyeballs for exophthalmos, strabismus, nystagmus and asthenopia.

Artificially produced conjunctivitis is to be looked for, because abroad we read of ipecac powder put into the conjunctival sac to produce it. In this country similar effects have been produced by silver nitrate, copper sulphate and pepper. Lunar caustic has been used to produce scarring of the cornea, though in the latter

instance the peculiar gray color is pathognomonic of the intent. Reports from Russia speak of the use of the needle for similar purposes.

Oblique focal illumination will further reveal the condition of the cornea.

The ophthalmoscope is also to be used to determine the condition of the lens and the fundus.

Suppose the conscript feigns total blindness in one eye: We have two valuable objective tests—the position of the visual axes, and the pupillary reaction.

If one eye turns up and out it is pretty good evidence that the eye is useless, and vice versa.

The pupillary reaction depends on the fact that in the dark the pupil dilates and in the light it contracts. The reflex arc is the optic nerve as the afferent nerve to the primary optic centers and the efferent nerve is the third cranial. The examinee is placed before a well lighted window and the examiner covers the eyes with his hands, cautioning the examinee to keep looking into the distance. After a few seconds he removes one hand and observes the pupillary reaction. If the pupil promptly contracts, there is sight, and the more prompt the reaction, the better the vision is likely to be. Of course, there are few exceptions to this rule. Synechiae prevent prompt reaction. The same is true of paralytic mydriasis.

The flashing of a bright light suddenly into the eyes is also a valuable means of eliciting not only this reaction but in the majority of instances will also produce flinching if sight is present. The eyelids will close spasmodically.

The consensual reaction is also to be obtained, that is, the behavior of the other pupil when one eye only is uncovered, the other remaining covered. For, with vision in the covered eye reaction consensually takes place, provided the reflex arc is intact, and vice versa. In every instance avoid touching the patient's face. In every instance the examinee is to look into the distance to avoid using accommodation and convergence with their attendant pupillary reactions. Of course, the ability of the examinee to converge, as well as his power of accommodation, is to be noted.

The behavior of the eye under a prism of eight centradis placed base out is interesting. To secure binocular vision the eye will turn in, and vice versa. (Priestly Smith and Edward Jackson.)

Place before the good eye a prism of six or eight centrad base out. The eye will move in and the other eye if blind will move in the same direction, and to the same extent. If binocular vision exists the alleged blind eye will not move at all. (Jackson.)

Have the examinee read; and while reading press the finger tip on lower lid of the supposed blind eye, displacing it and if really blind the reading will continue uninterrupted. If it sees, confusion ensues for diplopia is produced.

There are a number of subjective tests used to detect malingerers and it behooves the examiner to be acquainted with a considerable number of them in order not to run the same test with all as shrewd malingerers are liable to trade information received thus and make it the more difficult to detect subsequent ones. In all these tests the examiner must sit directly in front of the examinee or on the side of the alleged blind eye, and keep his gaze on it at all times, for the malingerer may attempt to close the eye and thus defeat the test for malingering. On several occasions I detected that trick and used the incident to browbeat the examinee into a confession of trickery and thus secured sight in an eye alleged to be blind.

The principal tests are as follows: I shall not enumerate all, time forbids:

Duane's Test.—Place on a white piece of paper some letters, alternately in red and black. Place over the good eye the ordinary red glass and ask him to name the letters or to read. If he reads all he sees with the alleged blind eye. If the other eye were really blind he would see only the letters in black.

Michand's Color Test.—Upon a white background are printed the usual Snellen letters. But some of the letters have additional strokes of red so that an O is converted into a Q, or an L into an E or a P into a B. A red glass is placed before the good eye and if the letters are read as modified as above, vision exists in both eyes.

Snellen's Test.—In a frame is placed alternately red and green glass of such density that when one piece of each is placed over the other, no light is transmitted. On these pieces are blocked off the letters according to the Snellen scale, thus giving a chance to get the acuity of the examinee's distant vision while testing for malingering. Before his eyes is placed a pair of specs in one cell of which is a piece of the red glass and in the other a piece of the green glass, and he is asked to read the letters. If he reads all he is not blind at all. This frame must be exposed only in testing. If he is really blind in one eye he will see the letters of one color only.

Cuignet's Test.—Cause the examinee to read aloud some small printed matter and while thus reading place a lead pencil between the paper and the face, holding the pencil vertically. If he reads without hesitation he is not blind in one eye. A modification of this test is the Javal-Cuignet shoe box test.

Cuignet's Test with a Lighted Candle.—Hold a lighted candle before the examinee's good eye and then slowly move it to a position in front of the alleged blind eye, or better to its outer side. If he says he still sees it he sees it with the alleged blind eye for his nose will cut off vision with the good eye.

Graefe-Baudry Test.—Take precaution that you are concerned with the good eye only, slyly covering the alleged blind eye with the hand. Place over the good eye a prism of sixteen centrads base up so that monocular diplopia is produced, and which he acknowledges. Now slowly push it up at the same time uncovering the alleged blind eye. If he still sees double he lies as to its condition and is not blind in it at all.

Duane's Test for Close Vision.—Have the examinee read out loud some small printed matter and while thus engaged slip before the alleged blind eye a prism of four centrads, base up. If he reads with confusion he is not blind at all in that eye.

Rendering the Examinee Artificially Myopic.—Place before the good eye a 6 diopter lens, small print can only be read at 17 c.m. with that eye. At this distance hold some test type and ask the examinee to read and while thus engaged slowly withdraw the card. If he still reads he reads with the alleged blind eye. Harlan's test is but a modification of this test as he uses a 20 diopter lens, convex or concave before the good eye.

Jackson's Test.—Place before the good eye two strong cylindrical lenses, one convex and one concave so that they neutralize each other. Have the examinee read and while thus engaged turn one of the cylinders so that it sets crossed with the other. If he still reads he reads with the alleged blind eye.

Todd's Test.—Place before the good eye a black disk with a pin hole in its center. The examinee is asked to read aloud and while reading thus his head is tilted backward until the line of vision is thrown below the hole in the disk, and the letters are no longer visible with that eye. If he reads the letters still he reads with the alleged blind eye.

Todd's Test with the Worth Amblyoscope.—The amblyoscope is set so that the objects placed in the distal ends of the tubes appear crossed as, for example, the bird and cage. It is then locked in this position and carelessly laid on the table of the examiner. The instrument is placed before the eyes of the examinee and he is asked to state what he sees with his right eye, and what with the left eye, of course watching that he does not close either eye. If the bird is in the right tube and he sees it with the alleged blind left eye he prevaricates.

The 6 or 8 Diopter Cylinder.—Make some vertical parallel lines on a piece of paper and cross them with horizontal parallel

lines so as to form squares. Place before one eye a 6 diopter cylinder axis 90 degrees and before the other eye a 6 diopter cylinder axis 180 degrees. Hold the paper thus ruled about 3 feet from the eyes. If he sees squares he sees with both eyes (Kugel). This test has been modified so that before the good eye is placed a 6 diopter lens axis 90 degrees, and the examinee is asked to look at lines ruled vertically on paper held 3 feet away. If he counts the lines he sees with both eyes.

The Double Prism.—Place before the good eye a double prism horizontally. If he sees three lights ahead while looking at the candle he sees with both eyes. If he says he cannot see two lights he lies.

Friedenburg Test.—The principle of this apparatus is to reflect into a mirror into which the examinee looks illiterate test type. The cards of type are placed in racks at the sides of the eyes in the general device; ahead in the frame held in the hand is the mirror which can be swung into position before either eye. It is further pivoted so that the examinee cannot tell which eye is being tested. A fair test of visual acuity is also possible.

Harman's Diaphragm Test.—While this instrument was not invented for this purpose yet it is valuable to detect malingering. It is the reverse of the bar reading test. Instead of a bar the examinee negotiates a screen in which there is a hole. Through this he looks at the test card 14 inches away. If he can read all the letters on the card he sees with both eyes.

Berthold's Test.—Take a prism of 20 centrads and hold it before the alleged blind eye, while the examinee is reading aloud some test type of the Jaeger scale. If good vision exists it will be very difficult to do so.

Nettleship's Test.—Put in the good eye a drop of a 1 per cent. solution of atropin and bandage the eyes for one hour. Then place in the hands of the examinee some reading matter, uncover the eyes and ask him to read. If he does so he sees with the alleged blind eye.

PRETENDED TOTAL BINOCULAR BLINDNESS

Schmidt and Rimpler Test.—Ask the examinee to put up his hand before his face, and to look at it. The really blind knowing its position in space readily accomplishes the command. The malingerer will hesitate and look in different directions for it.

Van Welz's Test.—In a darkened room place in front of the examinee a lighted candle in such a way that he will naturally look in its direction. Place before one eye a prism of say 8 centrads base out. If vision exists in the eye it will turn in and vice versa.

The examiner may go to one side of a room interposing a barrier as he goes. Then ask the examinee to approach him. If he avoids the barrier, he sees it. Care must be taken to avoid accidents with the truly blind.

In a hospital, under close scrutiny, tightly bandage both eyes of the examinee. If he possesses vision he will reveal his true state very soon for it is very irksome to be thus blindfolded very long.

The Surprise Test.—Direct suddenly in the direction of the examinee's face your fist. If he sees he will flinch. Fallot suggested in 1836 that even if he withheld from shrinking, the man will be unable to prevent increased action of the heart. The value of this suggestion he probably obtained from Sir Walter Scott's stories of the sale of a horse supposed to be blind. "The bargain was nigh struck when a blind man who was present said the horse was blind, a statement verified on further examination. Asked how he knew, he replied that he passed one hand before the animal's eyes, and felt his heart with the other. There was no increased beating."

Uverricht tested a malingerer with milk colored red with anilin and placed it before him. The milk was left untasted.

The reaction of the pupils is to be noted as described above. It may give one a clue to the acuity of vision.

The really totally blind, moreover, have the peculiar hesitating gait, the expressionless face and the upturned eyes.

EAR TESTS

It is expected that the ordinary conversational voice will be used in making the tests that will determine the recruit's fitness for service. It ought to be heard at a distance of 20 feet. The record is made in the form of a fraction whose denominator is always 20 and whose numerator is the distance at which the voice is heard. One ear is tested out at a time. The other is tightly closed by pressing the tragus into the external auditory canal. The examinee stands exposing the open ear to the examiner. He is commanded to close his eyes. The test is begun a short distance away and he is asked to repeat whatever is said to him. We usually use the figures and numbers up to 99. He may hear perfectly say up to 10 feet and refuse to hear beyond that, and then it is best to step back quietly the full 20 feet and ask him in a low voice "do you hear me now." Very many will respond, "No, Doctor, I do not." Of course we command the clerk, "Mark him normal," for he has malingered.

Or, we may begin at 20 feet away and say to the examinee, "Raise your hand when you hear a spoken word." Then the examiner approaches him repeating the same word or number over and over. The simulator will in due time raise his hand when the examiner gets close to him. This demonstrates him to be a liar for he heard the command at the full 20 feet away.

At one place in our state the examiner lined up a number of

conscripts and stepping back 20 feet asked in a low voice that all who could not hear him well to step out of line. Four obeyed his request, exposing themselves thus to the charge of malingering. In an adjoining county the examiner was much perplexed by one attempting to deceive him as to the condition of his hearing. He happened to remember that he owed the examinee a \$5 bill. Stepping back the full distance of 20 feet, he said in a low voice just above a whisper, "Come around to-night and I will give you that \$5 I have owed you for some time." To his uttermost surprise he replied, "All right, I will be there." Such devices while not strictly scientific are otherwise to be commended because they save a lot of valuable time.

The tests usually used are as follows:

The Weber Test.—If a tuning fork in vibration is placed on the vertex of the examinee it will be heard best in the ear closed say by a finger pressing the tragus into the external auditory canal. The same condition obtains with stenosis of the eustachian tube. If placed on the vertex of a malingerer he will say that he hears it best in his alleged good ear. Now if that ear be closed he will say that he does not hear it any more. Of course, he lies because he ought to hear it best in the closed ear.

Kerrison Test.—In a patient who claimed to be deaf in one ear as the result of injury, the drum head appeared normal. He assumed a degree of deafness which only could have come from injury to the labyrinth. The caloric test was applied to the alleged deaf ear and the reaction was quick and normal in every respect. In the confusion ensuing on the vertigo produced, it was very evident that the unilateral deafness was assumed.

Bárány Noise Apparatus.—This is one of the best of the recent tests. It is applied as follows: The examinee is asked to read aloud some printed matter and while thus engaged the noise apparatus is placed in the good ear and the noise started. If the examinee is really deaf he will raise his voice and at times such an one will fairly shout in order to hear himself. If the voice is not raised he is not deaf in the alleged deaf ear.

Confusion Test.—Take two rubber tubes and put on the end of each a funnel. The other ends are placed in the external auditory canals. Have two persons read dissimilarly into the funnel ends, one into each. If good hearing exists in both ears the examinee will be unable to repeat what is being read to him thus. It is best to have the reading done at his back. The examinee is to be blindfolded.

Chimani's Observations.—He recommends repeated tests with the acoumeter and the metronome. If slight differences exist in the different examinations the person is probably telling the truth; wide variations indicate malingering.

Erhard Test.—This test is based on the fact that the normal ear even when tightly closed will hear the tick of a loud watch.

The alleged deaf ear is closed and the loud ticking watch is approached to the ear acknowledged normal. He is asked to count the beats or ticks. It is best to test at a distance of 3 meters. The normal ear is now closed and the test is repeated, at a distance of 1 meter. If he says that he cannot hear the watch he is malingering.

Wagner Test.—This is the most recent one and comes to us from Chicago. The sound of a vibrating tuning fork is received into a funnel from the end of which runs a rubber tube to a bifurcation or a Y and from the latter run two rubber tubes on the ends of which are funnels meant to cover the examinee ears. The total length is from 6 to 7 feet. To conduct the test both ears are covered by the funnels and the examiner with an assistant stand behind the examinee. Each have in their hands tuning forks of the same pitch (C2). Both are set in vibration simultaneously, and one is placed on the vertex of the examinee and the assistant holds the prongs of his fork in the mouth of the distal funnel. Sound is thus transmitted both by air and bone conduction. Repeat thus two or three times. Then the examiner places his tuning fork, now still, on the vertex while at the same time the assistant places his sounding fork in the mouth of the funnel. The tube leading to the good ear is pinched shut and if the examinee says he still hears the fork he malingerers.

Stenger Test.—Two A tuning forks are used in this test; both are set in vibration, and one is held to each open ear. They are now moved back and forth so that only one fork can be heard at a time. This is repeated several times and confusion will result because he will after awhile be unable to tell with what ear he is hearing. If he says that he cannot hear the tuning fork in the good ear when he should, he malingerers.

Distraction of Attention.—Chauvigny found it a good procedure in testing a malingerer with tuning forks, to distract his attention and after awhile by dropping his voice the malingerer was able to hear.

SIMULATED BILATERAL DEAFNESS

Persons assuming such a rôle will have great difficulty in playing the part. Inconsistencies of conduct will crop out too easily. They overdo matters. A close watch should be put over such subjects. Inquiries among their associates will likely reveal their true condition.

The countenance of the totally deaf is usually passive. They are suspicious that persons are talking about them when in a crowd. They are rather downcast, quite in contrast with the totally blind. The lines of the face run down. They are pessimistic as a rule.

If a labyrinthine lesion is the cause of the deafness the

caloric, galvanic and rotation tests will all be negative; there will be no nystagmus, no vertigo.

The method of surprise may reveal the true state, like dropping behind such a person some coins which he will turn around to pick up perhaps, or turn around when some insulting remark is made or when his name is called, or if he be invited out for a drink if of a bibulous disposition. The test of Chauvigny outlined above may help.

Of course, differentiation is to be made between hysterical deafness and that of malingering.

It will be expected that direct examination of the ears will be made with the speculum and mirror to determine the condition of the external auditory canal as well as of the membrana tympani.

With the tests outlined above the surgeon will be able to quickly detect malingering in the great majority of cases. There are a few that may baffle the surgeon for a time, but truth will out.

DISCUSSION.

DR. A. KREBS, Pittsburgh: Several years ago I was asked to study two especially difficult cases of alleged monocular blindness, and in preparing the cases for court, I made a thorough study of the subject of simulated blindness and was surprised at the multiplicity of tests for the detection of malingerers that I found in the literature. If I remember correctly, I found over 100 tests or their modification. While a knowledge of all these known tests is no doubt desirable, it is seldom necessary to employ more than a few, with the use of which it is possible to become perfectly familiar.

As the essayist has stated, all malingerers are untruthful, and I might add that it is frequently necessary for the examining oculist to be even more so.

In my experience I have found it to be of the utmost importance to obtain the confidence of the patient. I have thus been able in some cases to make the claimant see the hopelessness of his contention that his sight had been impaired, and have been able to secure his confession that he had been malingering. It has been my custom in examining these cases to proceed just as I do in cases requiring correction of their errors of refraction. After obtaining the history as thoroughly and sympathetically as possible, I determine the vision as admitted by the patient. I then proceed with the routine examination of the external eye, muscle balance, pupillary reflexes, transparency of the media and appearance of the fundi. I then, as a basis for future tests, carefully determine the errors of refraction present by means of retinoscopy without the use of a cycloplegic. Tests are now made to see if improved vision results from the use of the indicated test lenses. If necessary, the visual fields are now taken as thoroughly as conditions call for. As the result of the routine examination, positive evidence of malingering may be found; if not, if further evidence is desired, the various tests for malingering are not made, the information previously obtained enabling me to judge as to which might prove of most value in a given case. Should the first

examination prove unsatisfactory, it is repeated as often as necessary or possible. For obvious reasons, cycloplegics are not used in these cases, except with the full consent of the patient.

Among the numerous tests recommended, I have found a convenient and rather satisfactory one in Fridenberg's instrument. Many of you are no doubt familiar with it. I show it for the benefit of those members who are not. The mirror reflects a test card in such a way that it is seen by the opposite eye, which eye the patient believes to be unconcerned in visual act.

Another test that I have not seen described in the literature that I employed with success in one subject is the following: Two 14 degree prisms were placed base out before the eyes. In looking at a well illuminated white card 12 inches square placed across the room against a dark background, fusion not resulting, crossed diplopia is produced. Because of the spectroscopic effect of the strong prisms, each card is seen with a bluish margin on the side corresponding to the apex of the prism through which it is seen, and a reddish margin opposite the base. Therefore, when the patient stated that he saw only the card with the blue margin on the right side and the red margin on the left, he admitted that he was seeing with his alleged blind left eye. Had fusion taken place, the movements of the eyes would of course also have proven the presence of sight in this eye, as in the prism tests of Priestly Smith and Edward Jackson.

So much has been written on the subject of ocular malingering, both from its medical and its legal aspect, that I feared nothing new could be presented in a paper of this kind. I therefore congratulate the essayist for being able to add still more to this interesting branch of ophthalmic knowledge.

As regards the malingering of those drafted for examination as to fitness for service in the new National Army, I am glad to say that, of almost 1,000 examined by me, I have found no malingerers nor any evidence that any member of our profession has been so unpatriotic as to suggest anything of this kind to any candidate. On the contrary, I have had more difficulty in obtaining exemption for those with eyesight below the requirements, since some of the examining boards feared that they were being imposed on and gave themselves the benefit of the doubt. Where possible, the staff of these examiners should include an oculist, because all doubtful cases could at once be examined with the ophthalmoscope, a method which enabled our board to make prompt and reliable decisions.

DR. LINN EMERSON, New York City: In this matter of malingering, I think it quite important that we should be fair to the applicant as well as to be on the lookout for malingering.

My observation of a large number of boards of examination, leads me to think that the attitude of the draft board is that of the district attorney, i. e.: the prisoner is presumably guilty.

One of my patients had 4 D. of myopic astigmatism; he had a dead labyrinth on one side and heard a watch in the better ear at 1 inch. He came to me asking for a certificate and I said: "No, you will have no difficulty." Much to my astonishment he was accepted. One of his draft board with whom I communicated by telephone said: "He could hear an ordinary voice at 10 feet, so we accepted him." Meanwhile the man, whose mother was dependent on him, lost his job before being returned from camp as unfit.

It is unfortunate that so few of the boards have oculists. Several boards have sent their applicants to me asking that I examine and give

a report. But many other boards in my locality have examined and passed men that I know will be sent back, and meanwhile they have lost their positions, and they will come back and be on the lookout for jobs.

One man who was referred to me claimed to be blind in one eye. I elicited from the man the fact that he had been examined for glasses by another oculist six months before. I learned from this oculist that the patient had 20/15 vision in each eye at that time. I put one-half per cent. cocain in the alleged bad eye and homatropin in the good eye, after which with a — .25 S. over the homatropized eye, and a + .25 S. over the cocainized eye, he read Jaeger No. 1, proving conclusively that he had good vision in the presumably blind eye. I was particular that he should have no chance to close either eye.

DR. A. HAYDEN, Chicago: Dr. Keiper's paper is timely just now, when all are doing some of this work of examination. I feel, however, that the malingering in the districts where I have examined has been very slight. I have examined 1,500 men for the new Army, and out of that number only three were under suspicion at all. It is quite a different proposition in the aviation section of the signal corps, where the men are particularly anxious to serve. We have to reverse the process and be very careful that they do not memorize the test cards and appear to possess more vision than they have. This is true in taking the pass pointing tests. We blindfold them to keep them from opening an eye a little. I think Dr. Emerson's suggestion that they put men on these boards to make routine examinations is an excellent one. I want to place before you a little scheme which I will show later in regard to hearing, and it works also in taking the vision and economizes time. The board in Chicago examined 600 men at a session of a few days; with one clerk and one assistant I was able to examine all these eyes and ears myself. A number of men, about fifteen, would be marched into the room; the clerk seated at the desk and the men backed against the wall on the opposite side from the test card. Then all made to face the wall. Start at one end of line, with finger in left ear first and then the right and make the test, and then come back plugging up the ears, and turn each man around and face the test card. We would not have them read a whole line, but one or two letters, so they could not learn them from hearing the other men read them. In that way an enormous number of men can be rapidly examined, and the time saving element is all important. Any suspected of malingering would be set aside and reexamined at the end. If there was any question at all they were sent to the office of some oculist for a very detailed examination.

MAJOR W. B. LANCASTER, Boston: I endorse heartily the suggestion made that the general attitude of the examiner should be sympathetic and friendly rather than domineering and overbearing. The object is not to make him read against his will but to lead him to betray himself unwittingly.

I think it an advantage to have test letters and test objects in different parts of the room and at different distances. The idea is to confuse the examinee by asking him to read different charts at different distances. If his visual acuity varies more than is reasonable we are justified in thinking he is not playing fair. Instead of starting him at the top of the Snellen card, I find it an advantage to expose only those letters that will pass him. Many of the men who attempt malingering are of a rather low grade of intelligence and will read the top line or two lines whether this means V 20/200 or 20/40. They have been told perhaps to read only the first two lines and no more. If the first two lines

are 20/200 and 20/100 they are rejected, but if we cover the upper part of the chart with a smaller chart which leaves exposed only 20/40 and under, the man will not infrequently read the top line or even two lines giving $V = 20/40$ or $20/30$. Simple and childish as this method seems, experience has shown its frequent value.

It is an advantage to point to the letters and make the man give some answer. The most difficult cases are the stolid Slavic type who claim they cannot understand English and cannot read or write. You should insist on some answer. If when tested on the Snellen E, they say it points in exactly the opposite direction to the correct one and get it exactly reversed every time they probably see it, this shows the advantage of insisting on some answer. The fact that we were somewhat limited in time, having a good many to examine in a short time, made it necessary to arrive rather quickly at a decision.

The ophthalmoscopic examination is of the greatest importance. It should never be omitted, in fact, it is the first thing to do.

Sometimes the test that we used was to throw a coin on the floor, it is very hard for the man not to follow it with his gaze and see it stop. Watch his eyes as he does that. Tell him to pick it up, and if he claims he has no idea where it is and his efforts to show that he does not are ludicrously exaggerated, he is convicted. Once I said quickly, "Heads or tails?" exposing a coin on the table about 3 feet from the man. He answered correctly the first time and wrong every time afterwards, the consistency with which he gave the wrong answer proved that he was seeing just as conclusively as giving the right answer would do. He could not get it wrong *every* time unless he saw it. One should avoid manifesting surprise, on the contrary you should appear to believe everything the man says, thus leading him on. One of my colleagues, Major Casey Wood, had the men walk barefoot where there were obstacles. It was amusing. He also scattered on the floor some of those rubber tacks, which look so much like metal tacks.

MAJOR ALLEN GREENWOOD, Boston: About two weeks ago I went to five of the southern cantonments to inspect the base hospitals, and the question of the malingerer as appearing before the examining boards interested me. They seem to send almost any man who comes before the draft board into the drafted army and many of them have to be rejected later. I found records of six men with glass eyes who had been passed by the draft boards. As the army does not take men with but one eye those men were sent home and the Government had been put to the expense of sending them, and the men to the expense of giving up their work. I saw in two or three of the hospitals several cases of absolute imbecility sent down. One had the typical appearance of a Mongolian idiot. They have found in the examination of recruits many strange anomalies, such as men with six fingers, men with three or four testicles and thousands of deformities. As regards malingerers, it requires a great deal of careful study to detect these cases. About the lowest percentage of rejections I found in any cantonment was $8\frac{1}{2}$ per cent. The rejection boards have various methods of examination. The weeding out of the unfit men is exceedingly interesting.

If you are anywhere near any of the cantonments it is worth your while to make a visit.

DR. A. H. ANDREWS, Chicago: It is unfortunate we did not hear the rest of the paper, which apparently was going to tell of malingerers with regard to the ear. One test is the stethoscopic test. A long, soft rubber tube stethoscope should be used, the examiner behind the

patient, the tubes inserted in the ear, the examiner's finger ready to grasp the tube that is inserted in the ear acknowledged to hear, and suddenly cut off the tube while reading the numbers. Say 416, and suddenly shut off the tube in the sound ear; the patient will say 400, leaving off the 16 if he does not hear in that ear. Another test is the perforated plug. Plug up the good ear and he does not hear you. Then use another plug which is perforated without his knowledge, and he thinks you have plugged up the good ear and he won't hear a word. It is easy to detect he is a liar, but to what extent is difficult. The other test is the tuning fork test. Let us assume that this is a very large fork of very low pitch. (Dr. Krebs acting the part of a patient.) Assume the left ear to be deaf. He hears in the right ear but not in the left. The handle of the fork is pressed into the normal ear and he is shut off. I ask if he hears, and he claims he does, and that means he hears in the ear he claims to be deaf. Pierce of Chicago is the first I have heard mention it. I have used it many times with good results.

DR. E. MAYER, New York City: I can answer some of Dr. Emerson's objections by stating that in the new draft there will be advisory boards of seven men, and as the men are rejected or accepted that claim rejection, they may go before these advisory boards composed of specialists and they will be accepted if they are found to be acceptable. That has already been done, and I have seen the paper and had something to do with drawing it up.

DR. KEIPER (closing discussion): In order to have covered the subject I should have read the paper entirely. There are a couple of newspaper reporters here who have asked me for a copy of this paper for publication in their papers. This I have declined to give as the public has no business to become acquainted with the manner of making these tests. Malingerers will become more adept in the art of deception thereby and thus harder to detect. I would suggest that information of the kind contained in this paper be kept *sub rosa* by us. In making these examinations we are to be in the attitude of kindly judges and not austere prosecutors, and it is surprising how easily we make these examinations thus, by being affable and joke with these men under examination. The number of these tests is legion and the vast number is liable to embarrass one as to which ones to use. I have attempted to pick out those which are most available and which have stood the test that experience brings, besides being available to the general practitioner. I have been conducting these examinations for the pension bureau for the last twenty-six years in the capacity of "expert examiner," and I wish to assure you we run up against some shrewd ones who wish to get more pension than what they deserve. For the detection of unilateral deafness, I wish to emphasize the test made with the Bárány noise apparatus. The noise apparatus is placed in the good ear and started. If the examinee is really deaf in the other ear, he will fairly shout when asked to read aloud the printed page placed before him. Dr. Andrews has mentioned the test with the binaural stethoscope. Permit me to call attention to its modification by Wagner of Chicago. One of the most valuable tests for malingering deafness is the Stenger test.

The surprise test of throwing coins on the floor back of the examinee tests both hearing and sight, and is thus doubly valuable. Kerrison's observation is valuable and attention is directed to it in the part of the paper unread.

Remember this—no matter how closely a normal ear is closed it still possesses some hearing. This observation becomes valuable as a basis for the tests mentioned for detection or malingering. It is a big job

to go over these conscripts, and one must be vigilant. When a defect appears in sight or hearing one must be cautious to take plenty of time in making the examination. Ofttimes it is best to set such an one aside for future examination, preferably by some one else.

Since writing this paper, there has appeared a book on Malingering by Jones and Llewellyn which deals with this subject from all of its angles, and is very interesting reading.

LATENT NYSTAGMUS

MAX W. JACOBS, M.D.

ST. LOUIS

During the past forty-five years occasional cases of nystagmus have been reported which are characterized by the fact that the essential eye phenomenon appears only when one eye is covered or binocular vision interfered with. The eyes of some of these patients did show occasional nystagmoid movements with both eyes open but in such the nystagmus was intensified by covering one of them. Fromaget has suggested calling this condition "latent nystagmus," because of its analogy to latent strabismus. Dorff¹, in 1914, described several cases which come in this category and has reviewed the literature up to that time. With the newer methods of ear examination doubtless some of the reported cases could have been excluded from this classification but the more recently examined patients have enjoyed the benefit of such newer methods and are reported as otologically negative. Disturbances of motility, especially convergent strabismus, are frequent in these cases and not rarely some form of nystagmus has been found in the ascendents and descendents.

My patient is a boy, aged 14, who consulted an oculist because of occasional poor vision when reading.

V.O.D.=18/70 to 18/40. Ophthalmometer shows R and L+.50 D. astigmatism x.90 V.O.S.=18/200 to 18/100.

Binocular vision = 18/20 (+).

There is no strabismus. The patient has been seen repeatedly and occasionally nystagmoid movements have been detected when both eyes are uncovered but, on the other hand, there have been visits to the oculist during which the boy was subjected to repeated examinations and during which such movements have been absent. On covering either eye marked horizontal nystagmus occurs. The excursions are of equal length and duration. The phenomenon is most marked in the left eye and both eyes almost immediately become quiet again when the obstruction is taken away. Retinoscopy after using homatropin shows 1 diopter of hypermetropia with no astigmatism. With the corresponding correction there is, of course, practically no change in vision. General examination and a blood Wassermann are negative. The otologist after making labyrinthine tests reports the ears normal and the eyegrounds show no abnormalities.

1. Dorff, H.: *Klin. Monatsbl. f. Augenheilk.*, October-December, 1914.

As Dorff¹ has stated it is assumed today that there is a supranuclear tonic association center which controls the ocular movements associated with binocular seeing. The center must be connected with the cortex, cerebellum, vestibular apparatus and nuclei of the eye muscles. Coppez assumes that there is a rhythmic or clonic center from which arise nystagmoid movements. This clonic is ordinarily controlled by the tonic center. In my patient, as in the first case described by Dorff, no peripheral cause for the disturbance in equilibrium could be found and I believe we can assume that we are dealing with an underdeveloped tonic association center which does not sufficiently inhibit the clonic center.

Fromaget tried to explain latent nystagmus on the theory that the tonic center which controls normally all eye movements requires the stimulation of both retinae to retain control over the antagonistic clonic center. My observations make me side with Dorff who pointed out that interference with the vision of one eye by means of a strong convex lens did not prevent the appearance of nystagmus. Both retinae were subjected in this experiment to a light stimulus and yet the nystagmus appeared. I have frequently held a screen in front of one eye of my patient in such a way that a reasonable amount of light was admitted to that eye (the other eye fixing). The horizontal nystagmus appeared immediately. These facts it seems to me make more tenable the suggestion of Dorff that in these patients, most of whom are strabismic, some fusion stimulus is still transmitted to the tonic association center and tends to maintain equilibrium. The placing of an obstruction before either eye even if it does not cut off all light, interferes with even this minimal fusion and the tonic loses control of the clonic center. This theory receives even stronger support in my case because the boy does not squint.

It has been suggested that we can be of service to these patients, particularly when we see them during childhood, by advising against occupations which require monocular vision. One of Dorff's patients noted his affliction when he first tried to fire a gun. As soon as he closed one eye to aim, the target became indistinct. Another patient was suddenly given work to do which required monocular vision for the greater part, and found that she was unable to see objects clearly under such conditions. Operations for strabismus have been of cosmetic value only. Stereoscopic exercises to strengthen binocular fixation have been suggested by Fromaget for those who still retain good binocular vision. The loss of an eye in one of these patients would bring

up the question whether greater damages should be assessed than after enucleation in a patient not suffering from this anomaly. The entire subject is still largely theoretical and the solution of its problems depends on our acquiring a better knowledge of the centers which control motility.

DISCUSSION

DR. JOHN GREEN JR., St. Louis: We are indebted to Dr. Jacobs for drawing our attention to this interesting variety of nystagmus. I believe this is the first case to be reported in this country.

Latent nystagmus has to be differentiated from nystagmus of otic origin, that due to organic nervous disease, that associated with congenital or early acquired amblyopia, miner's nystagmus, and finally, nystagmus dependent on hereditary syphilis, attention to which has recently been directed by Igersheimer. Although the diagnosis of latent nystagmus may seem perfectly obvious when we find a patient developing a horizontal nystagmus on screening one eye, every effort should be made to determine the presence (or absence) of any of the etiologic factors just alluded to. Failure to secure otologic and neurologic data throws doubt on the genuineness of some of the cases cited from the literature by Dorff. We should remember that we are traveling in the realms of pure hypothesis when we speak of a supranuclear tonic association center in control of a clonic center which is assumed to govern nystagmic movements. We are forced, however, to believe that some such mechanism does exist, and that through the failure, permanent or transitory, of the higher center to inhibit the lower center, nystagmus develops. We may yet find that the transitory nystagmus of labyrinthine origin and latent nystagmus are alike dependent on the same influence causing a momentary disarrangement in the delicate nervous mechanism.

In most of the reported cases the patient noticed something wrong only when circumstances compelled him to use one eye to the exclusion of the other. When we recall how rarely in ordinary visual tasks an individual abandons binocular for monocular vision, it seems likely that there may be many people with latent nystagmus who have never been incommoded by their trouble and hence have never sought ophthalmic advice. We all know how many people there are with one eye amblyopic who reach adult life without discovering their defect; it is only when by chance monocular vision with the amblyopic eye is momentarily substituted for binocular vision that the defect is brought to the attention of the patient. In like manner the existence of a latent strabismus often remains unknown.

Now that our attention has been directed to latent nystagmus I am sure that we shall all be on the lookout for it and that American ophthalmic literature will soon contain reports of cases of this interesting anomaly.

DR. E. E. BLAAUW, Buffalo: I rise to ask a few questions. How do you make out the binocular vision, and how do you say: "there is 18/20"? I infer there is true stereoscopic vision.

I think cases of nystagmus will be found more often, if looked for; slight cases will easily be detected during ophthalmoscopy in the upright image.

DR. V. A. CHAPMAN, Milwaukee: What I wished to say has just been alluded to. I believe this latent nystagmus is due to interference with stereoscopic vision and is an attempt of the single eye to establish stereoscopic vision. I would like to ask the essayist if he tested this

eye by using a tube in front of this single eye to see if the nystagmus would stop. I believe that method is used by artists in looking at paintings to bring out the stereoscopic effect. I believe it would stop the nystagmus if a tube 6 inches long were placed before this eye. I would like to know if that was tried.

DR. JACOBS (closing discussion): I did not use the tube, but I shall be very glad to try it if I can get hold of that patient again. He happens to be a youthful criminal who has escaped from the reformatory, and for this reason some of the tests I would have liked to make were not made. I tried to get hold of the child to make some additional examinations. I realize this report is not as full as it might be as to details.

As regards stereoscopic vision, his vision was merely taken with the two eyes open, reading from a test card. That was the only test made at any time. The boy read 18/20 plus from the card with both eyes open, and as soon as one eye was closed the vision sank very noticeably. That is as definitely as I can answer the question.

THE HYPOPHYSIS CEREBRI AND ITS MORPHOLOGIC INFLUENCE

F. PARK LEWIS, M.D.

BUFFALO, N. Y.

It is the purpose of this paper to direct attention to the glimmering of what may ultimately become an illuminating physiologic and pathologic fact. The demonstrations are not yet sufficiently clear to serve as a basis on which to predicate such a conclusion, but physiologic experimentation and clinical observations both point so strongly to the principle about to be formulated that there can be no question whatever as to the desirability of extending the inquiries in order that the range of its application may be more fully understood and its limitations determined. It is this:

That the hypophysis cerebri when normally functioning exercises a controlling influence not only over the skeletal and muscular structures, but over the nutrition and development as well as over all of the tissues having an epiblastic and mesoblastic origin.

If this conclusion is correct its bearings must have a far reaching significance. It will then follow that its aberrations of function will find their expression not only in frank manifestations of acromegaly, and in the less commonly recognized forms of dispituitarism which have been observed, but in a wide range of atrophies and dystrophies which we have been obliged to relegate vaguely to congenital deficiencies or other equally indefinite causes.

It will also be found, indeed it is already known, that between the advanced forms of hyperpituitary activity manifesting itself in the young subject in gigantism, and in dispituitarism in retarded development, both physical and mental, in those cases in which the secretion of the gland is temporarily or permanently limited, or its character altered, is the entire gamut of possibilities in which vital physical disturbances occur without there being of necessity any discoverable alteration either in the organ itself or in the bony cavity in which it rests.

The development of the skeleton is regulated by internal secretions. The development of the genital tract and its acces-

sory glands is subject, in the male, to the action of the interstitial glands, and in the female, to that of the corpora lutea, which also regulate the development of the breasts. Finally, the development of the germinative gland depends, at least in part, on the action of the thyroid, as well as of the central nervous system. We have no idea of the nature of the phenomena which takes place in any of these organs, under the influence of the secretions mentioned. We may say that they are phenomena of assimilation, but that does not advance us at all.

Let me briefly enumerate the recognized conditions which are present when the inhibitory control—whether it rests in the posterior portion of the gland, in the thyroid, the parathyroid or elsewhere—is released, and the full force of this dominant organ is brought into play, there are not only skeletal changes, but the integument becomes thickened, the skin glands become more active, hair grows abnormally over the body, the sexual appetite may at first be increased and then lost, glycosuria, or polyuria, are present. These are vital, fundamental physical changes that could come only through an effect produced on the embryologic tissues from which they originate. There are no known drugs that can cause all of these results. There are no known diseases, except those originating in the endocrinal glands that can so influence the primordial cells.

DEVELOPMENT OF THE PITUITARY BODY

The pituitary body is the final development of one of the first, and doubtless most important, of the embryologic tissues of the notochord. It is a small organ about the size of a shelled hazel-nut and weighs only a little more than 0.5 gm. The anterior—the glandular portion which dominates the growth of structures—grows from the invagination of the buccopharyngeal epithelium. The posterior portion is an extension of the infundibulum and comes from the midbrain—from the floor of the third ventricle. Vestigia of the buccal ectoderm sometimes persist and are known as the pharyngeal hypophysis. In the dural lining of the sella neuroglial elements are found which are called parahypophysis. Both, it is thought, may, in the event of the major structures being destroyed, take on their functions. As Cushing observes:

“Curiously enough, the anterior lobe of buccal origin receives its vascular supply from a number of small arterioles which pass down the infundibular stalk, whereas the posterior, or infundibular lobe, is nourished by a single artery which enters from below and behind.

"Now the effect of the active principle which has been obtained only from the pars posterior on plain muscle fiber is definite and specific. Like the extract of the suprarenals it produces a great rise of the blood pressure, with a contraction of the vessels and increase in force of the heart beats."

"Moreover," according to Schafer, "while adrenalin either produces no action on the coronary vessels, or in some cases causes them to dilate, the autocoid of the pituitary constricts them as it does most other systemic arterioles."

It has further been shown that the extracts of the posterior lobe also contain a substance which stimulates the flow of cerebro-spinal fluid.

The only analogy which we possess to these conditions is the effect produced on the chromosomes in the ovum by the addition of the ferment introduced by the sperm. In order that we may understand, even to the limited degree to which it is known, the normal and the perverted action of the hypophysis, we must know that of the other glands of the group; for rarely, if ever, is the function of one of them increased or suspended without the others being modified at the same time. As thyroid hypertrophy and overactivity produces the syndrome known as tachycardia exophthalmica, with proptosis and the other well recognized symptoms, so does a deficient secretion give rise to degenerative changes no less characteristic, while in the former the rapidity of the bodily metabolism is greatly increased, the heart action quickened, and the nutritive changes so hastened that there is a consequent loss of weight, perspiration is easily induced, and a tendency exists to the production of glycosuria.

HYPOTHYROIDISM

A thyroid insufficiency is shown by the opposite of each of these signs; the bodily temperature is reduced and is usually sub-normal, the circulation is poor, the pulse rate reduced, there is greatly increased sugar tolerance, the skin is dry, rough and eczematous. With this comes obesity, an expressionless face, carious teeth, gingivitis, or actual pyorrhea. In children the sexual development is delayed—immature sexual organs only being present in many cases—the stature remains stunted, and there is a faulty mental development. The effect of an atrophy of the thyroid in young children is most marked. As Schafer states:

"The muscles are limp and weak. Deafmutism is common. The highest functions of the nervous system remain undeveloped.

the child becoming idiotic. This seems to be due to an arrest of development of the cortex cerebri."

All of this is important because on removal of the thyroid the pituitary undergoes enlargement and gives every indication of increased secretion. When the thyroid secretion is excessive there is an increased elimination of calcium from the body, and per contra with a thyroid deficiency there is a retention of these salts, resulting in atheromatous formations in the walls of the arteries and in calcareous degenerations elsewhere. As the activity of the thyroid decreases with advancing years it is not unlikely that the development of cataract may be hastened thereby. With these facts in view the statement first predicated may be carried a step further by saying:

That any interference, mechanical or otherwise, with the normal functioning of the hypophysis will exercise a controlling influence on the metabolism, the growth and development in the young subject, and on the nutrition and normality in the mature of all of the structures within the sphere of its activities.

We cannot, therefore, ignore in this connection the action of the other ductless glands. Gigantism is produced, not only by overaction of the pituitary, but as Bramwell has shown, it is associated also with sexual development, and increased stature is a result of castration before puberty. With dispituitarism comes atrophy of the ovaries and testes so definitely that the relationship is incontestible. The diminutive stature of cretinism shows that the thyroids and parathyroids have also an influence on elemental cellular structure, while in hypopituitarism, the sub-normal temperature, the slow pulse, the low blood pressure, and the sluggish mentality give a clinical picture closely resembling the myxedema associated with thyroid insufficiency.

We have, then, in the series of ductless glands interacting ferments controlling cellular activities which are seemingly as potent as those which are developed in the elemental cell. There is no structure which escapes. With the enlargement of the bones of the face, with the modifications of the skin, and with the change in nerve structure, the fibrous tissues are at the same time involved. And, when activity of any of these glands is disturbed and the balance of function is lost, metamorphic changes may be expected although they may manifest themselves only in the smallest degree. Morphogenetic substances and hormones act in very minute doses; they are substances

which appear to act after the fashion of a nervous excitation or of a diastatic action; they bring no energy to the tissues or organs which they influence, but they only liberate preexisting energy; they regulate anabolism and katabolism.

It has already been shown that the action of the pituitary is to contract the arteries. The pathology of retinitis pigmentosa is that of a slow atrophic process with invasion by the pigment epithelium of the atrophic retina. In some cases the vessels are so reduced in size that they appear as white lines and in others very dark lines can be discerned down the center of the pale path. Now, when this is associated with those forms of physical development which would indicate an overaction of the anterior portion of the hypophysis, and a reduction of the action of the posterior portion, the condition which we would expect to find is thus described by Cobb. He says:

"With one part of the gland functioning adequately, and the other deficient, the clinical picture might correspond to what is expected when the particular part of the gland is abnormal. Supposing the anterior part of the gland to be normal and the posterior deficient, we should expect a fully grown individual, but with excessive deposits of fat, slow circulation, sluggish mentality and the other features of hypopituitarism, with the exception of stunted skeletal development."

I have the good fortune of being able to present precisely such a picture as that just described.

This is a lad, aged 12, who is 5 feet, 4 inches in height. He weighs 120 pounds. The clothes which he requires are those of a youth of 18 years. He has a smooth skin; sexual organs quite undeveloped; he has a drooping mouth, which is usually open; a slow pulse; a well developed sella with a pituitary apparently of normal size; a developing retinitis pigmentosa; markedly atrophic arteries, with the characteristic narrowing of the field.

And if the contention is correct, so forcefully urged by Jones, that the excessive or deficient action of the pituitary may modify the development of the eye structures, we should expect to find in advanced forms of hyperpituitarism changes in the nutrition of the globe. Unfortunately, I do not find in a very large number of cases examined that these changes have been noted. Even so careful an observer as Cushing merely notes in certain of his cases "that such a degree of visual acuity was secured when corrected" without mentioning the character of the correction employed.

It has been my good fortune to follow with very close attention a case of acromegaly since the year 1902. The character-

istics are pathognomonic. The bitemporal hemianopsia follows an almost directly vertical line through the macula on one side and invading the macula on the other. When first observed there was an axial myopia of 10 diopters in each eye. This has progressively increased until it has reached 15 diopters, but under glandular feeding all of the symptoms, except the limited field and the increased myopia, have been markedly relieved. The especial interest in this case is the length of time in which it has been under observation, the typical syndrome which it presents, the verification of hypophyseal hypertrophy by the roentgenogram, and the conclusions to which the demonstrable changes give warrant.

REPORT OF CASE

History.—Miss M., who subsequently married, came under my observation in 1903. There was no suspicion at that time of there being anything other than the rather high degree of myopia, so that the general physical observations, which are not usually made by an ophthalmologist, were not recorded. There was nothing sufficiently characteristic in the facies to suggest the presence of the beginnings of a serious organic disease, although a narrowing of the temporal fields was already present.

The patient was a rather slight woman, aged 34, and about 5 feet, 6 inches in height. Earlier pictures show very little of the coarseness of expression in the face which subsequently developed. She has light brown hair of moderately heavy texture, rather full, somewhat protuberant eyes and a clear skin with a pink flush in the cheeks. She had worn glasses for the correction of her myopia since she was a little girl. At that time she was having some discomfort in using her eyes with an occasional sense of blurring.

Examination.—Her refraction under careful test was:

R—10.0 Dsph \ominus —0.50 Dcyl ax 150° v = 20/30
L—10.0 Dsph \ominus —0.50 Dcyl ax 180° v = 20/30

There was a choroidal atrophy around the disc with some pigmentary degenerative changes and a general fulness of the choroidal vessels without any marked staphyloma posticum.

Two years later, in 1905, the blurring was more frequent. Headaches had developed. The pain in the head began with scintillating motion on the temporal side; the pain usually being on the opposite side of the blurring eye. Vision was still 20/30 in each eye, with a slight change in the angle of the astigmatism, but as yet no increase in the myopia. At about this time a change in the facial expression must have begun, although it was so gradual that no definite date can be fixed. The following year, 1906, there was still no refractive change, but vision had become reduced in the right eye to 20/40 and in the left to 20/30. There were no further changes in the eye ground.

In 1907, a slight increase in the myopia was recorded. Vision had been reduced to 20/30 with difficulty in each eye. The temporal fields were still narrowing. By 1911, the myopia had increased in each eye to 11.50 D. Vision reduced in the right to 20/40, in the left to 20/50.

The next year the bitemporal hemianopsia had markedly increased. In the left eye vision was 20/100. The macula had been invaded, only on looking to one side of an object could it be seen. About this time an enlargement of the jaw was noticed. It began to develop a prognathous form. She was conscious that the bite was different from formerly. The nose became broad and prominent, and a peculiar symptom developed, which proved to be of importance in the subsequent study of this disease, and which the writer had not discovered in any of the observations connected with it. It is the opposite of the Stellwag sign in morbus Basedowii. In place of the upper lid being retracted, showing a white scleral line and imperfectly following the eyeball in its downward excursion the *lower lids droop, showing a wide scleral line from below up*. There was still a proptosis, but not of a sufficiently marked character to attract notice, but the lower lids seemed to sag down as though the nerve control was diminished.

Medicinal Treatment.—At about this period glandular feeding was begun. The face had assumed a somewhat myxedematous character, and the treatment given was largely the substance of the thyroid and thymus glands and of the corpus luteum. Once or twice the substance of the pituitary was given carefully and experimentally, but with an aggravation of the symptoms.

Results.—By 1914, there was an increase of the myopia, also a distinct increase in the visual acuity. The refractive values were as follows:

R —13.00 D \ominus —1.50 D cyl ax 105 \vee = 20/30
L —14.00 D \ominus —1.50 D cyl ax 75 \vee = 20/40

The visual fields on each side were limited by the macula, marked staphyloma posticum with a large crescent on the nasal side of each disc, but the headaches had wholly disappeared. The sense of blurring was also gone, and with definite bitemporal hemianopsia large objects could be discovered when moved through the obscured areas.

Present Condition.—In March, 1917, fourteen years after the first observation, the patient, now a woman aged 48, had a slight enlargement of the right lobe of the thyroid. Menstruation ceased at about the age of 37, but for a long time there was much irregularity in the menstrual functions. She has rather an excessive growth of hair on the face. All of the acromegalic symptoms, except that of height, are pronounced. The skin is clear, and at times she is subject to excessive perspiration, which rolls off her face. Attacks of dizziness, to which she has been subject, have disappeared. Urinalysis shows nothing abnormal.

Polyuria is present. She likes sweets, but not excessively. Her digestion is good, her rheumatism is much better than formerly. Her fingers are square and chubby. The field of vision taken on the tangent screen shows typical hemianopsia almost directly vertical reaching to the macula in the right eye and including the macula to the extent of 2 mm. in the left.

From what has been written we might expect the symptoms produced by overaction of the anterior portion of the hypophysis might be corrected by the exhibition of the substance of the thyroid, and a sufficiently large number of cases are now on record in which enlargement of the restricted visual field, improvement in visual acuity and general physical betterment have been noted. Particularly striking are the cases reported by De Schweinitz (*Archives of Ophthalmology*, March, 1917).

ORGANOTHERAPY

In addition to the substance of the thyroid, that of the thymus, has been of value. As hyperpituitarism causes a lengthening, a thickening and a strengthening of the bones, per contra the hypofunction of the thymus renders the bones shorter, thinner and more fragile. As increased pituitarism is associated with, if not the direct cause of the atrophy of the testes and ovaries and the organism is deprived of the internal secretion of these glands, the employment of their substance in the absence of any active principle obtained from them is especially justifiable. But the use of any of these materials causes at times profound effects and they should be used with judgment and caution. The initial dose should be small, and Cobb is undoubtedly correct in saying that these substances, especially that of the thyroid, are often given in doses that are too large.

In a paper presented before the New York Stomatological Society in 1902, and in another read before the American Society of Orthodontists in 1903, on the "Conformation of the Face in Relation to the Development of the Eye," I urged the importance of broadening the narrow jaw and removing lymphoid obstruction in the pharynx, in order that the lymph and nerve channels should be unimpeded, thereby preventing abnormalities in the area of distribution with the resultant atrophies, neuralgias and other trophic disturbances of the parts involved. It was then urged that the marked benefit that follows adenectomies and the opening of obstructed nasal passages was not wholly dependent on a freer admission of oxygen, necessary as this is, because there may be occlusion of the nose, without any such group of symptoms as adenoids presents. The relief comes from the

removal of the pressure on the nasopharyngeal vessels connecting with the hypophysis, the interference with the function of which causes an abnormal hebetude. Quite recently orthodontists are recognizing the importance of their work in this respect in the effect of the relaxation of constricted tissues on physical growth.

There are two other ways at least in which the activity of the endocrinal glands may be modified. The first is by excessive emotion, and there are many such cases in which this is the exciting cause. The second has been urged by Mr. Lane in his work on the "Colon," and is toxic absorption.

Dr. Zentmayer, in his address before the Section on Ophthalmology of the American Medical Association in June, 1917, found authority for ascribing to the disturbed action of the endocrinal glands hereditary optic nerve atrophy, optic atrophy with hyperthyroidism, an especial syndrome as described by Lamb, pigmentary degeneration of the retina as urged by Jones, retinal degenerative changes with osteitis deformans and amaurotic family idiocy.

Surely with such basic observations the subject is well worth the closest study until our knowledge of the relation of the ductless glands to organic eye changes is put on a more exact foundation.

HYPOPHYSEAL TUMOR CAUSING FEW NEIGHBORHOOD SYMPTOMS BUT MARKED BITEMPORAL CONSTRICTION OF VISUAL FIELDS

ACROMEGALIC CHANGES OF UNUSUAL DEGREE. SELLAR DECOMPRESSION. IMPROVEMENT

EDWARD STIEREN, M.D.,

PITTSBURGH

History.—April 16, 1917. S. A. S., bookkeeper, single, age 33, Bulgarian by birth. Referred by an optician for examination of his eyes, impaired vision being his chief complaint, but is concerned about acromegalic changes and some disturbance in mental functions.

States that the enlargement of head, feet and hands began about seven years ago; sight had been failing for about six months. The accompanying photographs (Fig. 1) were taken nine years ago, five years ago and at present, showing the marked changes that have taken place between each period.



Figure 1.

Fig. 1.—A, the patient as he looked nine years previously; B, his appearance five years ago, and C, his present appearance.

Ophthalmic Examination.—R. V. 6/20—; Homatropin: R. V. 6/30 with + 0.87 S. = V. 6/20. L. V. 6/6—; Homatropin: L. V. 6/30 + with + 1 S. \subset + .25 Cyl. 90° = V. 6/5. 1° right hyperphoria; 2° exophoria.

Fields: Right; temporal hemianopsia almost complete and extending almost to fixation. Left; temporal hemianopsia ex-

tending within 10° of fixation and irregularly encroaching on nasal side as well. Fundi negative. Media clear.

Physical Examination.—Made by Dr. C. R. Jones. Height 5 feet $6\frac{1}{2}$ in., weight 178 pounds.

Blood pressure: systolic 120, diastolic 70.

Urinalysis: specific gravity 1,033, acid, heavy flocculent sediment, trace of albumin, sugar none. The microscope revealed calcium oxalate crystals, few leukocytes, few epithelial cells.

Blood count: red blood cells, 4,740,000; hemoglobin 63 per cent.; white blood cells 5,800. Wassermann test negative.

Roentgen-ray Examination.—Made by Dr. C. N. Schaeffer. It revealed enormous enlargement of the sella with thinning of the floor and recession of the posterior clinoid process.



Figure 2.

Fig. 2.—Roentgenogram showing enormous enlargement of the sella.

Patient notes that his memory is not as good as formerly and that his sexual power is failing. No headaches.

Was seen by Dr. Lyndon H. Landon May 21. Sellar decompression was decided on and was accordingly performed June 2 at the Western Pennsylvania Hospital.

OPERATION. TRANSPHENOIDAL HYPOPHYSOTOMY.

Local anesthesia 10 per cent. cocain in 1-1,000 adrenalin solution. Mucous membrane of the septum on both sides was anesthetized for 20 minutes after which a complete submucous resection of the nasal septum was carried out back to and includ-

ing the rostrum of the vomer. When the latter was removed, the small remaining space of the sphenoidal cavity was opened. This cavity was nearly obliterated by the depression of the sellar floor. The opening was enlarged as far as possible in all directions and the sellar floor opened and enlarged in like manner as far as possible. Very little distinct capsule was visible covering the contents of the sella. A considerable amount of the growth was removed and preserved for pathologic examination after which the two layers of mucous membrane were permitted to fall together.

A grooved director was introduced between them into the sella and both nasal cavities were packed with bismuth albolene gauze, and the patient taken to the roentgen-ray department for determining wherein the sella was open.

The patient stood the first part of the operation very well but on account of the extreme difficulty in performing the submucous resection, the anesthesia was discontinued and toward the end of the operation some pain was experienced and also considerable hyperexcitability. A general anesthetic, however, was unnecessary.

Because of the extreme deflexions of the septum, it was impossible to do a resection without tearing the mucous membrane.

On the whole, the patient stood the operation very well and left the table in good condition.

Examination of the roentgenogram showed the grooved director passing into the sella and through the tumor tissue. No conception of the extent of the tumor could be obtained by this approach.

Dr. Landon's report:

"Further postoperative notes of this case show that the operation was performed on June 2. Packing was removed from the nose on June 3 and daily thereafter the nasal cavities were cleansed with adrenalin, followed by spray of Dobell's solution, followed by insufflation of oily spray consisting of camphor, menthol and eucalyptol in liquid albolene. The patient made an uneventful operative recovery.

I also enclose herewith a clinical chart which shows the almost complete absence of any postoperative rise in temperature or other complications. On June 8, the patient stated to me that his vision, subjectively, was improved; that he could read much better and with rough finger test, I found what appeared to be considerable widening of the field of vision. Pathologic report of the tissues removed from the sella show the growth to be carcinoma. The patient was discharged from the hospital on June 13, with free nasal breathing and no intranasal complications."

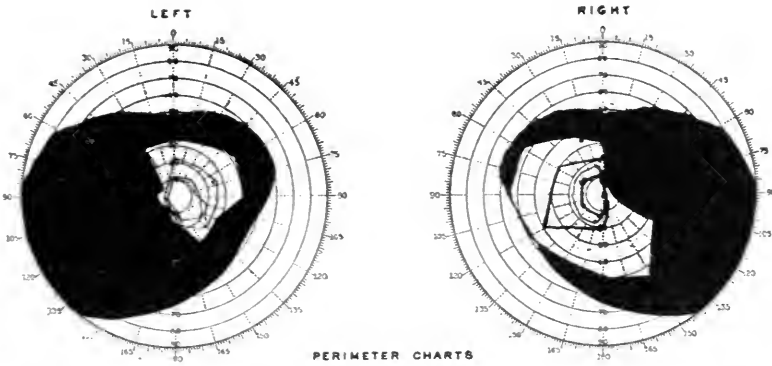


Fig. 3.—Perimeter chart showing the field of vision before operation.

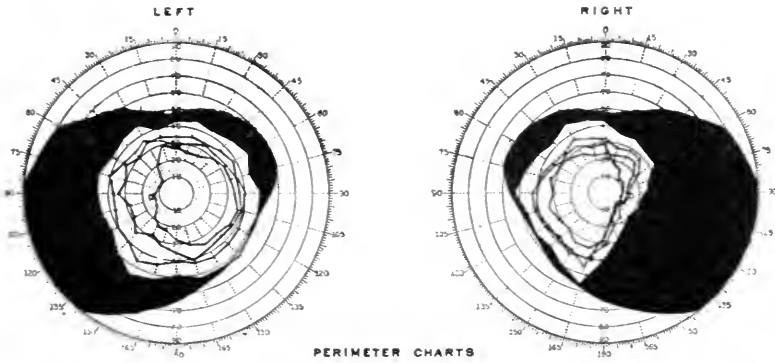


Fig. 4.—Field of vision after operation.

The fields of vision before and after operation (Figs. 3 and 4) show an enlargement after operation in all directions both for form and color. Patient's general health has improved and he has gained about 12 pounds. There is improvement in his mental condition in regard to memory, the sexual condition remains about the same.

DEEP ROENTGEN-RAY THERAPY IN THE TREATMENT OF TUMORS OF THE HYPOPHYSIS

CLARENCE LOEB, A.M.; M.D.

CHICAGO

Hypophyseal tumors manifest themselves by symptoms that are referable either to pressure, such as visual disturbances, or to interference with the endocrinal secretion, such as acromegaly, or by symptoms of a more general character such as headache and dizziness which may be due to either pressure or secretory disturbances, or perhaps both. It is unnecessary to go into the details of these symptoms, as they are well known to all of you. I will merely mention that the characteristic ocular symptom is a bitemporal hemianopsia caused by pressure posteriorly on the decussating fibers of the optic nerves. This is accompanied by more or less loss of central visual acuity, which in advanced cases causes a variation in the typical appearance of the fields.

It frequently happens that the visual disturbances are the first to cause the patient to consult a physician, and, therefore, the oculist may be the man to point to the correct diagnosis. But the correct diagnosis is after all not the point that interests the patient most. What he consults us for is to obtain relief from distressing symptoms, and the question that arises is, "What have we to offer him in the form of relief or, better still, cure?"

If the disease is a local manifestation of a constitutional condition, for example syphilis, we have, of course, the recognized specific therapy. If, however, our tests for these conditions prove negative, and if a roentgenogram shows an enlargement of the sella turcica, so that we may safely assume the presence of a neoplastic growth or possibly a cyst, medicines may alleviate or mask the symptoms but will not influence the course of the lesion. We must have recourse to some other form of treatment.

Naturally the question of surgical intervention presents itself. This may be in the form of a decompression, which is after all merely a palliative measure. Or an attempt may be made to directly attack and remove the tumor. There are various methods of approaching the hypophysis, but the best is probably the route suggested by Dr. Joseph C. Beck, namely, the transantral

or antrum-ethmoid-sphenoid route. However, it is not my purpose to discuss the various surgical methods; they are all difficult, and not devoid of danger, as the rather high mortality shows. Because a method of treatment is dangerous, however, is no argument against its employment in a condition which of itself is more dangerous to function and even life, unless some alternative can be suggested which will accomplish results as good with less untoward possibilities. Such an alternative I wish to present to you by detailing the course of a case of hypophyseal tumor treated by deep roentgen-ray therapy, which has been under my observation recently at the North Chicago Hospital.

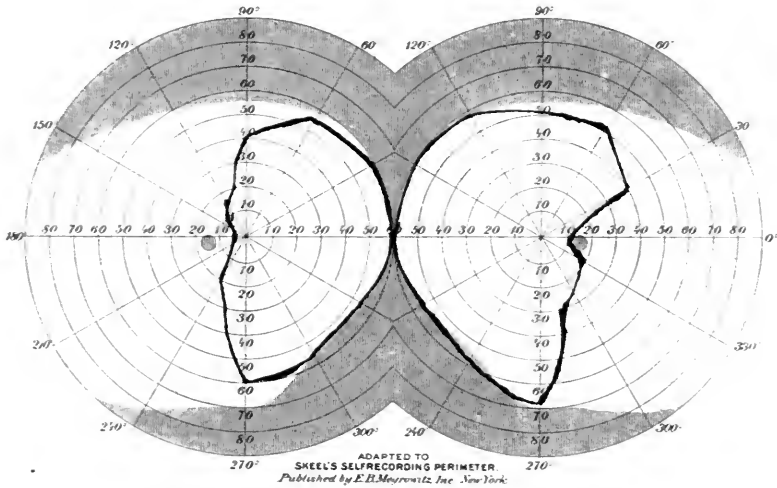


Fig. 1.—Fields of vision Mrs. H., Aug. 8, 1917.

Roentgen therapy of intracranial tumors is not especially new. In the furor which greeted the news of the discovery of the roentgen rays, and their exploitation as a panacea, their use in intracranial conditions could not be overlooked, especially as cranial surgery was then in its infancy. The usual number of marvelous results was announced, but sober second thought and a more careful scrutiny of cases caused the pendulum to swing in the opposite direction, until the use of the roentgen rays was practically abandoned. Contributing to its neglect was the fact that general surgeons such as Cushing, McArthur and Eiselberg, and especially otologists such as McKernon, Newman, and Beck showed that the brain under proper precautions was as safe for operative procedures as other parts of the body such as the abdomen and thorax, which careful asepsis and improved technic had added to the domain of successful surgery. If success had

invariably attended their efforts, and if it had been always possible to persuade the patient to submit to an operation, "finis" might have been written to this chapter of surgery. As it is, we must welcome the fact that there is a possibility of a recrudescence of roentgen-ray therapy for intracranial tumors, and must carefully investigate its claims to a place in our armamentarium.

In 1909, Gramegna¹ reported a case of acromegaly treated by roentgen-rays in which he obtained a temporary improvement in the visual fields accompanied by a complete disappearance of the headaches. In spite of treatment, however, the case went

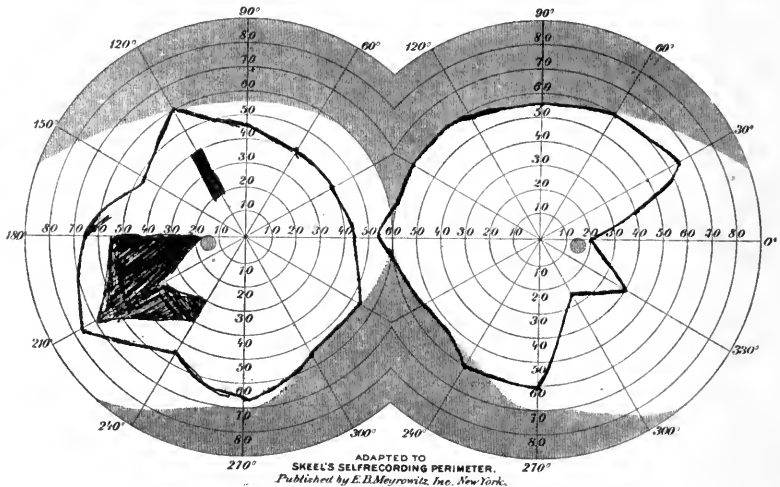


Fig. 2.—Fields of vision Mrs. H., Aug. 22, 1917.

on to a fatal termination. In 1913, Bécélère² reported four cases. The results of the treatment were as follows:

(a) Disappearance of headaches, dizziness, nausea and vomiting. Distinct improvement in vision, including increase in the visual fields, cessation of pathologic changes and restoration of the sexual function. The patient passed away from the direct observation of Bécélère, but the family physician reported that the patient was in good health and the same condition five years after treatment.

(b) Improvement in visual fields, also central vision. Some parts of the optic nerves, however, had already undergone degeneration. Status idem after one year.

(c) Increase in the visual fields and central vision. Impotence less. Status idem after one year.

(d) Slight increase in visual fields and central vision, complete disappearance of headaches. Still under observation.

Béclère's conclusions are that there are two classes of tumors from a symptomatic standpoint. First, those in which the symptoms are due to compression, especially of the optic nerves. These cases are the most favorable for treatment, and the roentgen-ray is useful in all stages, especially the early ones before atrophy of the nerves has developed. Second, those in which the symptoms are especially referred to the bony skeleton. In these, the abnormal growth can be checked, but the organic lesions already established cannot be made to disappear. In the early stages of this form, roentgen-ray therapy is indicated, but in the latter, in

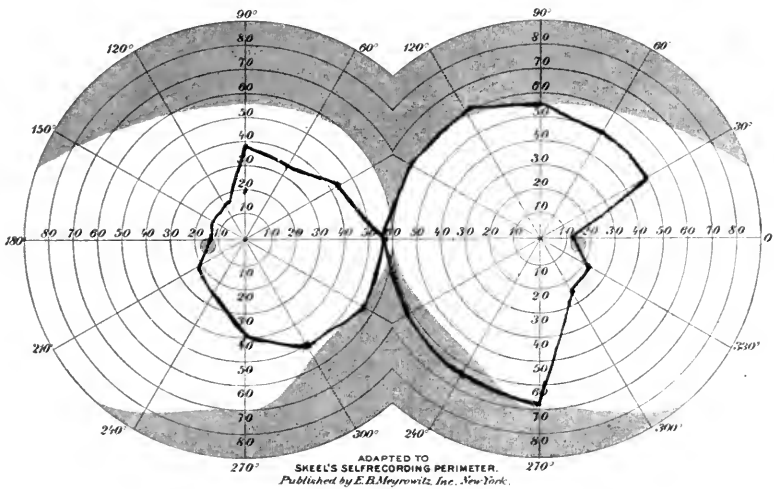


Fig. 3.—Fields of vision Mrs. H., Sept. 19, 1917.

which the hyperfunction has given place to insufficiency of secretion, it is contraindicated.

Other men have used the roentgen ray in the treatment of hypophyseal tumors, and their results have been quite satisfactory.

Calamet³ treated a case of typical pituitary tumor with roentgen therapy with only temporary improvement. The ultimate outcome of the case is not given.

Terrien⁴, however, reports a case of his own in which the improvement had lasted four years. He also refers to a case of De Lapersonne in which a distinct improvement took place in a case of acromegaly with disturbances in the visual field, headaches and enlargements of the sella turcica. Under roentgen therapy these symptoms became very much ameliorated. Cushing⁵ reports very favorable results from the use of roentgen

rays in several of his cases. Vision was restored after almost complete blindness by a combination of partial removal of the tumor and subsequent repeated exposure to the roentgen rays. He advises it especially after operation, because the roentgen rays thereby are able to attack the denuded lesion directly. He warns, however, that further experience in a larger number of cases may fail to substantiate the good results reported by earlier observers. It therefore becomes the duty of everyone who has had experience along this line to report his results, so that a definite conclusion may be made.

My own case is as follows:

History.—Mrs. H., aged 38, came complaining of headaches from which she had been suffering for five years. These varied

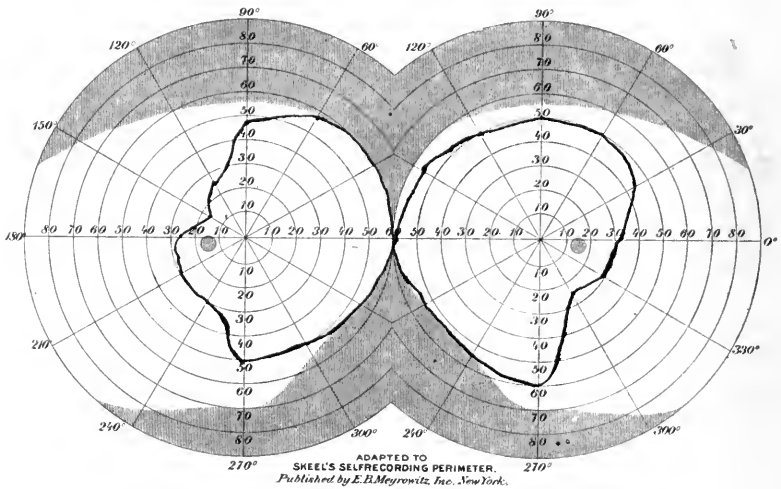


Fig. 4.—Fields of vision Mrs. H., Oct. 17, 1917.

in intensity but she was rarely free from them. In addition her sight had been failing for about the same length of time, and she had had many attacks of dizziness and nausea. When the headaches first appeared, she had been examined for glasses on the theory that her headaches were ocular, but had been told that she did not need glasses. Since then she had not consulted a physician. For about the same length of time she had noticed that there was a change in her appearance, namely, that she had been getting fatter. Her face was larger, her hands were larger, and there had been an increase in the body fat. There had, however, been no loss of hair on the body, nor any decrease in sexual instinct, although her menopause had set in about the same time, that is, at the age of 32, just after the birth of her youngest son. She had three children all alive and healthy, and her family history was negative. There had been no change in her voice. There was no increase in thirst. I may mention

here that the test for sugar tolerance was made impossible by the fact that the patient vomited when it was tried.

Examination.—The physical examination of the patient showed the following: height, 5 feet, 4 inches; weight, 175 pounds. There was a general increase in the adipose tissue. The fingers tapered; the lips were thick and the cheeks were fat. Her features were coarse. The thyroid was not palpable. All reflexes were normal. The physical examination of the heart and lungs was negative. The maximum blood pressure was 110; the minimum was 80.

The nasal examination showed large inferior turbinates on both sides, with hypertrophy of the whole mucous membrane:



Fig. 5.—Roentgenogram head Mrs. H. showing distorted sella turcica.

also very pronounced pyorrhea and calcareous deposits with diseased teeth. The lips and tongue were large. It was found that the pharynx was highly reactive so that the pharyngeal structures could not be seen.

Examination of the eyes showed that the discs were paler than normal, but there was no actual atrophy. As the patient was illiterate and her mentality was somewhat low, either because of the disease or naturally, it was difficult to test her vision. It was determined, however, to be $\frac{1}{8}$ in each eye. With a plus 2.0 S. lens, the central vision was nearly $\frac{6}{20}$.

Taking of the visual fields was also difficult for the same reasons as mentioned above and also because it was difficult to compel the patient to keep her eyes fixed on the central mark. However, it can be seen by the fields reproduced (Figs. 1, 2, 3 and 4) that there is a distinct loss of the temporal fields, although not a complete loss. In addition there is some impairment of the nasal fields especially in the left.

A roentgenogram of the patient's head showed a decided increase in the area of the sella turcica as shown in Figure 5. A normal sella turcica is shown for comparison (Fig. 6).



Fig. 6.—Normal sella turcica.

The patient was referred to Dr. Eisen, roentgenologist of the North Chicago Hospital, who carried out the treatment as outlined in the accompanying table. From time to time the patient reported to me for examination. I have never been able to see any change in the discs. Almost from the beginning there was an alleviation of the patient's headaches. They did not disappear entirely, but there were intervals of complete relief with an occasional headache of less severity. Also she was able to sleep, either because of relief from pain or improvement in her general condition. A field of vision taken August 8 showed practically normal nasal fields, but practically no change in the

temporal fields. Vision had increased to about $1/6$ in each eye without glasses.

August 22, the patient came to be examined in a very cheerful mood. She had been free from headaches for about two weeks except for a slight one the day before. Also she stated that she had been sleeping well, and that her sight was much better. The vision in each eye was $6/30$. The visual fields showed an astonishing improvement. The right showed a normal nasal field with improvement upward and slightly downward. The left showed some loss in the nasal field, but a temporal field of about $2/3$ normal extent. There is, however, a scotoma of considerable size downward and outward and a small one upward and outward.

September 5, vision was $6/30$ plus. As it was a dark day, taking of the visual fields was postponed until the following day, but the patient did not return until September 19. She had had no treatment for a week, and stated that she had felt much worse for the last five days. Her headaches had returned, and

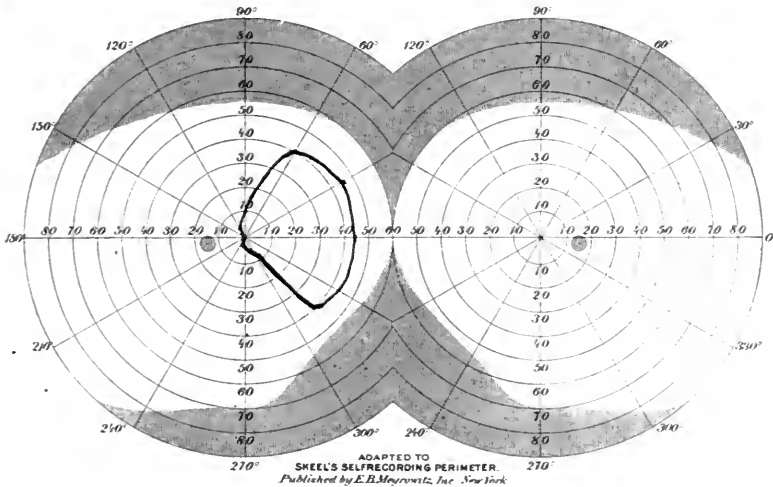


Fig. 7.—Fields of vision in case of Dr. Jos. Beck before operation.

her sight had grown worse. The vision was only $1/8$ and the visual fields were about the same as at the time of her first visit.

October 3, the patient stated that she still had occasional headaches and some nausea, but her vision had increased to $1/4$ without glasses.

October 8, the patient felt better and had no headaches. October 17, her general condition was still improving. Her vision was $1/4$ in each eye. The visual field showed some improvement over the last time. The discs were the same. On the whole, the improvement from the ocular standpoint had not kept pace with the improvement in headaches and general feeling.

Treatment.—The treatment in this case is given in detail in the subjoined table. Summarized, it was as follows:

SUMMARY OF THE ROENTGEN-RAY THERAPY EMPLOYED IN TREATING A CASE OF TUMOR OF THE HYPOPHYSIS

| Date 1917 | Skin Area | Skin Focus Distance in Inches | No. Treat. to Same Area = One Ery- thema Dose | Voltage or Spark Gap of Tube in Inches | Milliam- perage, Tube | Time in Minutes | Dose in Milliampere Minutes | Thickness Fil- ter of Alumi- num Besides Sole Leather |
|--------------|--|-------------------------------------|--|---|-----------------------------|-----------------------|-----------------------------------|--|
| 7/20 | One sq. in. over left temporal..... | 8 | 1 | 14 | 3/5 | 8 | 1 4/5 | 1 sheet |
| 23 | One sq. in. over left temporal..... | 8 | 1 | 12 | 1 | 4 | 4 | 1 sheet |
| 24 | One sq. in. over left temporal..... | 8 | 2 | 11 | 1 | 4 | 4 | 1 sheet |
| 25 | One sq. in. over left temporal..... | 8 | 3 | 12 | 3/5 | 5 | 3 | 1 sheet |
| 26 | One sq. in. over right temporal..... | 8 | 4 | 12 | 1 | 4 | 4 | 1 sheet |
| 30 | One sq. in. over right temporal..... | 8 | 1 | 8 | 1 | 4 | 4 | 2 sheets |
| 31 | One sq. in. over right temporal..... | 8 | 2 | 11 1/2 | 1 | 4 | 4 | 2 sheets |
| 8/1 | One sq. in. over right temporal..... | 8 | 3 | 11 | 1 | 4 | 4 | 2 sheets |
| 2 | One sq. in. over right temporal..... | 8 | 4 | 10 1/2 | 1 | 4 | 4 | 2 sheets |
| 3 | Two in. circle through nose..... | 8 | 1 | 10 | 1 | 4 | 4 | 1 sheet |
| 6 | Through lower tip nose..... | 8 | 2 | 11 | 1 | 4 | 4 | 1 sheet |
| 7 | Through lower tip nose (headache)..... | 8 | 3 | 12 | 1 | 4 | 4 | 1 sheet |
| 8 | Through lower tip nose..... | 8 | 4 | 10 1/2 | 3/5 | 4 | 4 | 1 sheet |
| 9 | Two in. circle, right antrum (headache) | 8 | 1 | 11 | 1 | 5 | 3 | 1 sheet |
| 13 | Two in. circle, right antrum (headache) | 8 | 2 | 12 | 1 | 5 | 5 | 1 sheet |
| 14 | Two in. circle, right antrum..... | 8 | 3 | 11 | 1 | 5 | 5 | 1 sheet |
| 15 | Two in. circle, right antrum..... | 8 | 4 | 11 | 1 | 4 | 4 | 1 sheet |
| 16 | Two in. circle, right antrum..... | 8 | 5 | 11 1/2 | 6/5 | 4 | 4 4/5 | 1 sheet |
| 21 | Two in. circle, left antrum..... | 8 | 1 | 10 1/2 | 3/5 | 5 | 3 | 1 sheet |
| 22 | Two in. circle, L. A., slight headache; cold | 8 | 2 | 11 | 3/5 | 5 | 3 | 1 sheet |
| 23 | Two in. circle, L. A., sleep and vision better | 8 | 3 | 9 | 9/5 | 4 | 7 1/5 | 1 sheet |
| 27 | Two in. circle, left antrum..... | 8 | 4 | 10 | 1 | 4 | 4 | 1 sheet |
| 28 | Two in. circle, left antrum..... | 8 | 1 | 11 | 1 | 4 | 4 | 1 sheet |
| 29 | Two in. circle, left temporal..... | 8 | 2 | 10 1/2 | 1 | 4 | 4 | 1 sheet |
| 9/4 | Two in. circle, left temporal..... | 8 | 3 | 11 | 4/5 | 4 | 4 | 1 sheet |
| 5 | Two in. circle, right temporal..... | 8 | 4 | 11 | 1 | 4 | 4 | 1 sheet |
| 6 | Two in. circle, right temporal..... | 8 | 1 | 12 | 1 | 5 | 5 | 1 sheet |
| 10 | Two in. circle, right temporal..... | 8 | 2 | 11 1/2 | 1 | 5 | 5 | 1 sheet |
| 11 | Two in. circle, right temporal..... | 8 | 3 | 12 | 1 | 5 | 5 | 1 sheet |
| 13 | Two in. circle, right temporal..... | 8 | 4 | 13 | 1 | 5 | 5 | 1 sheet |
| 20 | Two in. circle, eyes worse, headache 9/18 | 8 | 5 | 11 | 1 | 5 | 5 | 1 sheet |
| 24 | Two in. circle through nose..... | 8 | 1 | 11 | 1 | 4 | 4 | 1 sheet |
| 27 | Two in. circle through nose..... | 8 | 2 | 10 | 1 | 4 | 4 | 1 sheet |
| 10/1 | Two in. circle through nose..... | 8 | 3 | 13 1/2 | 3/5 | 4 | 3 3/5 | 1 sheet |
| 2 | Two in. circle, right antrum..... | 8 | 4 | 11 | 1 | 4 | 4 | 1 sheet |
| 3 | Two in. circle, right antrum..... | 8 | 1 | 13 1/2 | 1 | 4 | 4 | 1 sheet |
| 4 | Two in. circle, right antrum..... | 8 | 2 | 11 | 1 | 4 | 4 | 1 sheet |
| 8 | Two in. circle, right antrum..... | 8 | 3 | 11 1/2 | 1 | 4 | 4 | 1 sheet |
| 9 | Two in. circle, right antrum..... | 8 | 4 | 13 1/2 | 1 | 4 | 4 | 1 sheet |
| 10 | Two in. circle, right antrum..... | 8 | 5 | 11 1/2 | 4/5 | 4 | 4 | 1 sheet |
| 11 | Two in. circle, right antrum..... | 8 | 6 | 11 1/2 | 1 | 4 | 4 | 1 sheet |
| 15 | Two in. circle, left antrum..... | 8 | 1 | 11 | 1 | 4 | 4 | 1 sheet |
| 16 | Two in. circle, left antrum..... | 8 | 2 | 11 | 1 | 4 | 4 | 1 sheet |
| 17 | Two in. circle, left antrum..... | 8 | 3 | 10 | 1 | 4 | 4 | 1 sheet |
| 18 | Two in. circle, left antrum..... | 8 | 4 | 11 | 3/5 | 5 | 3 | 1 sheet |
| | Two in. circle, left antrum..... | 8 | 5 | 12 1/2 | 3/5 | 7 | 4 1/5 | 1 sheet |

The places where the rays were applied were those closest to the hypophysis, that is, over the right and left temple, right and left antrum, and nose. Between the tube and skin, an aluminum plate of 1 mm. thickness was interposed. The purpose of this was to cut out more of the "soft" rays, those of longer length, and concentrate the "hard" rays, that is, those shorter wave length, which are less easily absorbed by the tissues overlying the hypophysis.

The applications were made until an erythema dose had been given. This is a dose sufficient to produce a slight redness of the skin. In the beginning this was given in four applications of five minutes each in ten days to each area, covering the five mentioned areas in seven weeks. Lately the amount given is one-fifth less or four minutes, reaching an erythema dose in two weeks, or ten weeks for all five areas. The reason for this reduction is a clinical one, namely, the patient complained of nausea, dizziness, and prostration on the evening following the treatment. With the present amount these symptoms do not

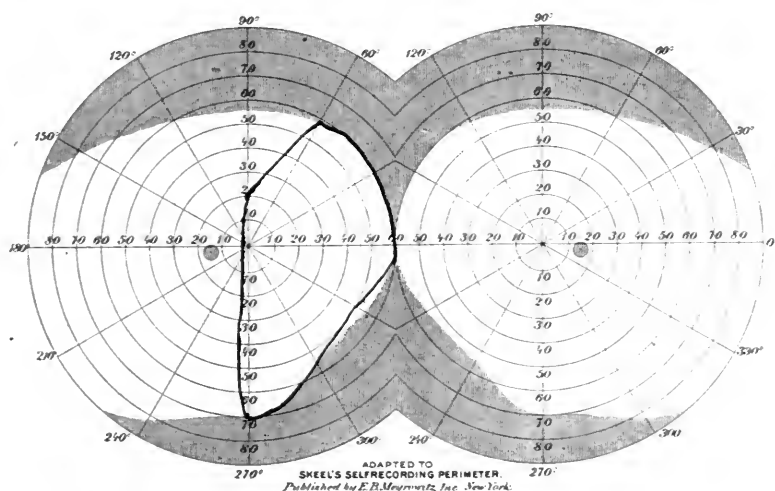


Fig. 8.—Fields of vision in Dr. Beck's case 3 years after operation.

appear. The skin-focus distance was 8 inches. The equivalent spark gap of the tube varied from 9 to 14 inches, usually 11 inches. From $3/5$ milliamperes to $14/5$ milliamperes were passed for four to six minutes, producing in each treatment from 3 to 7 milliampere-minutes. In all the patient has received ten erythema doses. The generator is a 16 inch coil with 110 volts, $3\frac{1}{2}$ amperes in primary, with mercury interrupter of 1,800 revolutions per minute; the tubes are water cooled and measure 27 inches from anode to kathode and 12 inches from kathode to automatic "Queen" reducer.

In contrast to this case, I should like to mention the result in a case of cyst of the hypophysis, that I recently saw. The symptoms date from the age of 13, some twenty-three years ago,

and consist of visual disturbances, lack of ambition, heavy smoking and drinking, headaches, skeletal, skin and adipose changes. Vision decreased to complete blindness in right eye (Fig. 7), with complete loss of the left temporal field, with contracted left nasal field, and great loss of central vision. Optic disk showed atrophy and a roentgenogram showed enlargement of the sella turcica. An operation by Dr. Joseph C. Beck revealed the presence of a cyst of the hypophysis, which was opened and drained. Recovery was uneventful. Three years later, the patient presented himself with cure of headaches and other symptoms. The skeletal changes had persisted but had not increased. Discs were pale. Vision R. E., perception to light, but inability to project. V. L. E. = $1/10$, no increase with lenses. The visual field for the left eye showed almost complete loss of the temporal field, but an increase in the nasal to almost normal (Fig. 8). The patient felt well, had good mentality, and on the whole was in a very satisfactory condition, considering how far the disease had progressed before the operation.

COMMENT

In conclusion permit me to compare the relative merits of the surgical and roentgen-ray treatments. Access to roentgen-ray apparatus is as a rule easy, whereas operators able to attack the hypophysis region are not so very numerous. The patient, unless otherwise incapacitated, is able to perform his daily occupation while under roentgen-ray treatment. Patients who refuse to undergo operation have a method of treatment which promises relief from symptoms and this has been known to last for four years or more. Whether that means a cure can of course only be proven by a postmortem. Decompression operations can do no better than that. Removal of the tumor may mean permanent cure, but this can always be held as a last resort in case the roentgen-ray treatment fails. It seems to me that the logical course is first to try whether or not the roentgen-ray treatment will prove of benefit. If the symptoms persist for a reasonable length of time, or if they become aggravated, then an operation becomes necessary, and this should be a direct attempt to remove the tumor if the patient is strong enough, otherwise a decompression followed later by extirpation of the tumor.

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DISCUSSION

DR. JOSEPH BECK, Chicago: The symposium itself covered a great part of the subject and yet a great many things were not touched on, particularly the diagnosis and the etiology of these conditions, therefore I shall say nothing about them in discussion but confine my remarks to the presentations. I wish to say that in the case presented by our chairman, which was a very interesting case with a splendid result, some observation should be had as to the changes in the hands and feet following operations. Dr. Sauer of St. Louis, one of our members, has shown us in his case that there was gradually a diminution in the size of the hands and feet, which was shown by placing the hands in a vessel of water, the displacement of the water becoming less and less. There was an appreciable recession in the growth. Also there is the question of feeding in these cases, which showed that the gland was probably destroyed by a cyst or a tumor. In regard to the case presented by my associate, Dr. Loeb, I reluctantly gave consent to roentgen-ray treatment because it was an excellent case for operation, and I think perhaps it will come to an operation later. However, the patient is remarkably well for the time she has been treated. In reference to the technic which interested me most, I would like to say that the subject has received the greatest attention by the rhinologist and the results proved that the best route is by way of the nose. I find it is more difficult by way of the septum than the method I advocate, namely, by way of the antrum. In the septum route as suggested by Hirsch the difficulties to be mentioned, are the heavy ledge of the septum and the thickness of bone at the sphenoid the difficulty in cutting through that is certainly a great deal more than in the side route through the antrum, which is shorter, and more direct vision is obtained, and the septum between the two sphenoids is easily broken down. My experience, however, is very limited in any type of operation on the sellar region.

DR. ARNOLD KNAPP, New York: Dr. Knapp stated that it was important to remember that in many cases the defect in the visual field was not the usual bitemporal one. He begged leave to quote the three following cases in this connection:

In the first case, the patient, a woman, was totally blind in one eye with optic atrophy. The other eye was normal and the field showed no anomaly even for colors. Symptoms of adiposa-genital dystrophy suggested roentgenogram which showed an enormous destruction of the sellar region. The patient was subjected to deep roentgen-ray treatment once a week for about six months, principally with a view to checking the headache. The results of this treatment were uncertain. They had no effect on the loss of sight in the left eye. The patient now (which is about four years since she first came under observation) is developing a contraction of the temporal field in her remaining eye.

In Case 2, another woman, there was a central scotoma in both eyes, a condition which in itself is unusual in women. On examining the color fields a distinct bitemporal achromatopsia was found. The roentgenogram then confirmed the diagnosis of a pituitary process.

The third case, a woman, presented one blind eye from optic atrophy. The other eye had normal vision and the field for white was normal, but on examining the color field in this good eye a beginning temporal defect in the color fields was found. The roentgen-ray examination confirmed the suspicion of a pituitary growth.

These cases were all women between 30 and 50 years of age, and he would like to close his remarks with the statement that in optic atrophy

in women the possibility of a pituitary process must always be considered.

DR. JESSE S. WYLER, Cincinnati: About two years ago Darier published a paper upon the roentgen-ray treatment of pituitary tumor, and while abstracting that article, a woman came into my office on whom I tried the roentgen-ray treatment. The woman reported to me Sept. 29, 1916. She was 40 years old and married. Her vision was 6/60 in the right eye and 6/36 in the left eye. The disc resembled a primary optic atrophy, a diagnosis which had been made by an oculist three years previously. Blood examination was negative and we eliminated every possible cause except that of pituitary tumor. The field was contracted for colors. The roentgenogram by Dr. Lange showed a very large sella turcica and acromegalic changes in the bones of the hand and feet. Now, after reading this article of Darier and not seeing any signs of inflammation or swelling in the optic nerve, I figured a decompression would not be advantageous. So I advised roentgen rays thoroughly applied and on November 15 she had received six treatments according to the method of Darier. One treatment a week. The cross-fire method in the temporal region was also utilized, because Darier reports that the mucous membrane in the mouth and nose had been badly burned. After these treatments the eye showed 6/36. Feb. 6, 1917, more treatments had been given, and the patient had 6/24 in the right and 6/36 in the left. The last time, on October 26, 3 days ago, and thirteen months after the first diagnosis she had no more treatments. The vision then was 6/24 in each eye. The result was not brilliant, still I think that in the light of what we have here accomplished, roentgen therapy might be tried more extensively in the future than it has been in the past.

DR. CLARENCE LOEB, St. Louis: When the secretary wrote asking me whom I should like to open the discussion I told him to get a roentgenologist. I enjoyed Major Johnson's speech. In Chicago we have a paper that publishes an article every week how to end the war and when I go back I will suggest that Major Johnson go over and give a speech to the soldier in the trenches; if the Germans hear the applause they will all come over to enjoy it. I agree with Dr. Lewis that there are more diseases of the ductless glands than we recognize, and I think the labor of the laboratory men should be very much increased along these lines and more delicate tests made either physiologic or chemical so we can recognize these conditions earlier. At the present time when we learn there is a disease of the ductless glands it is a very difficult thing to combat, whereas if we could get some symptom or some test that would let us recognize it in the early stages we would have better means to combat it. I do not feel my knowledge of operative procedures on the hypophysis would allow me to discuss it with Dr. Stein. I will leave that to the surgeon and the otolaryngologist. As to Dr. Knapp's cases, the report he made of them did not show on what he made a diagnosis of hypophysis condition. I must confess in my position near the window I could not hear everything he said. The only thing I got was that they did not present the typical hypophyseal symptoms of a bitemporal hemianopsia.

DR. F. PARK LEWIS (closing discussion): The time allowed for my paper made it necessary to confine it to the fundamental principles involved in the relation between the hypophysis and the other endocrinal glands. I did not take time to refer to three cases which have been under my observation during the past year. These cases, together with others which have been under the observation of my friends, lead me to believe that hypophyseal disturbances are far more common than we have thought. Undoubtedly such lesions are frequently overlooked, es-

pecially in the early stages before pathognomonic visual fields have been developed. It is no doubt important that we should know more definitely concerning the meaning of enlargement of the sella. It is entirely possible that these enlargements may be as much due to changes in growth as to pressure symptoms. A special symptom which I have noted and which may be characteristic in pituitary hypertrophy, is one which might be considered as the opposite to the well known Stellwag sign in hyperthyroidism. Instead of the white line of the sclera showing above I have noticed in pituitary enlargement a drooping of the lower lid with a lack of tonicity so that the white scleral line shows below. Much has been said concerning the value of radiant treatment. My experience leads me to approach it with great caution. In one of my cases in which it was used with full dosage in the intensified form for two treatments at intervals of one week the effect produced was pain and confusion in the head, physical depression and more rapid decrease of sight. It is quite possible that under other conditions it might be used more effectively but certainly for the present until we know more about the conditions for which it is appropriate it should be used with great caution.

THE RELATION OF RETINITIS PIGMENTOSA AND ALLIED ATROPHIES OF THE RETINA AND OPTIC NERVE TO THE DUCTLESS GLANDS

WITH REPORT OF CASE TREATED WITH THYROID EXTRACT

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Some time in the year 1910 there came to me, for considerably impaired vision in both eyes, not helped by glasses, a mulatto girl, aged 15. Ophthalmoscopic examination showed black spider splotches on the dark fundus natural to the negro, not readily visible at first inspection. This is the only case of retinitis pigmentosa I have seen in the colored race. Being mindful of the futility of treatment for this disease, as far as text books and literature went, I decided to strike out in some other direction. As thyroid extract had been proved in later years to have a marked influence on retarded mentality in certain states, as cretinism, and a mysterious influence over constructive metabolism, it was the agent selected. This view was further strengthened by the close relation of the optic nerve to the brain, giving rise to the expression that "the optic nerve is the finger of the brain." Unfortunately this case could not be followed up, as the patient removed shortly afterward to Baltimore, and could not be traced. (This patient reported, May, 1918, and holds all vision gained.)

The earliest case of which a record could be made is the following:

CASE 1.—C. E. J., man, aged 42, presented himself for examination Aug. 16, 1910. Sight known to be failing for several years, with both temporal fields moderately contracted, and partial night-blindness: RV=18/60—, Jaeg. No. 10—; LV=18/50—, Jaeg. No. 10—. No raise by glasses, far or near. Pupils medium size, not reacting to light, but doing so on convergence. Marked retinitis pigmentosa both eyes. Put on thyroid extract, 1 grain three times a day. Took one bottle of tablets, probably 100. As this patient did not return, a letter of inquiry was sent, to which he replied, Feb. 5, 1912, sending samples of fine print for close about Jaeg. No. 7 for each eye and 20/80+ for distance. He was located and induced to return, July 29, 1916; pupils revealed slight movement to light, dilate slowly in dark: RV=20/40; LV=20/50. Each with S + 4.0, Jaeg. No. 3; together

J. No. 2 easily. Fields almost full, except temporal ends; fundus same, slightly pigmented. Put back on 1 grain thyroid three times a day, to which was added $1/60$ grain strychnin and $1/30$ grain arsenic. The arsenic was added from a belief in its alterative value, and the strychnin as a bitter tonic, only indirectly beneficial, to build up lowered nerve tone. This formula has been in use for several years past, as will be seen in later cases. He was induced to return, Aug. 19, 1917, having taken four boxes of sixty pills each: RV=20/40 —, S + 4.0, J. 1 —; LV=20/50, S + 4.0, J. 1. Both eyes open V=20/40. Pupils fixed; fields probably a little more contracted on temporal sides. The notable feature is that the downward tendency of the disease, as far as this single case is concerned, was checked by the first treatment, covering only one month.

CASE 2.—Mrs. S. E. R., aged 50, came shortly after previous patient, complaining she could not see to read with any glasses, which fact was verified by tests. A record made at the time, and checked off on one or two return visits, was hopelessly misplaced, but fixed sufficiently in memory to be sure she could not read newspaper with either eye, and in all respects had a typical retinitis pigmentosa, with night-blindness. She was put on thyroid alone, and by request returned on June 3, 1917, volunteering the statement she knew her sight was better, best attainable being R = 20/75, J. 14 —; L = 20/50, J. 2. She has taken the tablets on frequent occasions during these years.

CASE 3.—D. H. H., aged 44, was examined Aug. 18, 1910. This patient is reported by reason of being an uncle of Case 4. and probably indicating the course of the disease in this particular family. Noticed first failing of sight twenty years ago, for night only. Seen by me in 1906 with clear diagnosis of retinitis pigmentosa. Previous to this he had been under the care of one of Philadelphia's leading oculists, receiving strychnin injections and electrical treatment. In 1906, V = 20/50 each eye, J. 3. Seen in 1908 with rupture of left cornea and traumatic cataract: RV = 20/80, S + 3.0, J. 6. Fields much smaller. Pupils dilate very slowly in the dark. Some parts of fundus entirely black. As an experiment, put on 2 grain doses of thyroid, t.i.d. Feb. 7, 1911: V = 18/120, S + 3.0, J. 12. Fields more contracted. Beginning cataract. Gave trial injection of cyanide of mercury with dionin subconjunctivally. Sept. 23, 1912: V = 9/200. J. 14. I think, and he believes, it is the cataract more than the retinal disease reducing vision. Put back on thyroids with arsenic in increasing doses, which he took until the heart was upset by the thyroid, and the stomach by the arsenic. Aug. 21, 1917, returned by request. Barely counts fingers; pupil almost opaque; light fields as large as five years ago. In view of the formation of cataract, which seems sclerosed, and permits a little light through by electric ophthalmoscope, it is not possible to draw any conclusion as to what the sight would be had no cataract formed.

CASE 4.—B. F. W., aged 28, Sept. 15, 1915. This patient is a nephew of Case 3. Night-blindness since 8 years old. Had been educated as a pharmacist and had secured a position in Philadelphia, where he took treatment from same oculist his uncle had. Realizing the gravity of his condition, he decided to give up his profession and return to rural life near his small home town. Has distinct atrophy of retina and optic nerve, contracted retinal vessels and very fine pigmentation visible only by direct examination, as fuzzy chopped up hair all over fundus. Vision in each eye varies to 20/50 + and J. 3 — to J. 1 —. Fields contracted to a 5 inch circle at 20 inches, with an acute indentation, fingerlike, on temporal sides. Put on 2 grains thyroid, and increasing doses Fowler's solution of arsenic three times a day. Feb. 22, 1916: RV = 20/40, J. 2 +; LV = 20/40, J. 1 clearer. Added strychnin and arsenic to thyroids in pill form. Tests made on Nov. 16, 1916, Jan. 31, 1917, and Aug. 27, 1917, all about same. Fields as large as at beginning; pupils do not react visibly, but on remaining in the dark quite a while, they enlarge as though a weak mydriatic had been used, and are correspondingly slow in returning in bright light. Vision in each eye is 20/40 readily, with some of 20/30, and J. 1 easily in each. The value of a slight raise in these cases is in showing arrest of downward tendency of disease.

CASE 5.—C. H., aged 10, was examined May 17, 1913. Convergent strabismus right eye; very poor use of left. Skull lopsided, teeth terraced in front, no enamel on tips. RV = fingers 4 feet; no Jeag.; LV = 18/160, J. 6-8. Dilated pupils; by shadow both hyperopic 7 D. No raise. Gave same for constant wear. Optic discs fibrous, retinal vessels white streaks with red centers; entire fundus studded with black spots like pepper, coarse and fine, thickest at periphery and more black than red area; sight worse in fading light; fields good; pulse 60; blood pressure 85-90. Put on 1 grain thyroid t.i.d. on alternate weeks. Dec. 23, 1913, pulse 100, blood pressure 85; V = 20/100 +, J. 4-3 —. Gets on in school better. In this case, the raise in vision may be due to wearing the glasses. Returned by request, Sept. 17, 1916. Took tablets only in 1913; LV = 20/75 +, J. 3; pulse 60, blood pressure 92; pupil active. Put on strychnin 1/100 grain, arsenic 1/50 grain, thyroid 1 grain t.i.d. Sept. 23, 1917: V = 20/50 +, J. 1 —. Retinal vessels at disc barely colored, soon becoming white threads. Choroidal vessels visible where pigment is not massed. Family history indicates hereditary specific infection. Sees better than formerly by dim lights.

CASE 6.—Mrs. S., aged 50, May 18, 1916. Came to get "good specs," so she could see; would not be convinced it was a case of bad eyes, although her father had been blind many years and several of his relatives blind or near-blind, some of them presumably from same disease. Noticed night-blindness before 20 years of age. Retinitis pigmentosa in both eyes of extreme degree. Pupils inactive to light and convergence. V. each 20/75 —, S. + 4, J. 3, each. Fields in circle of 1 foot in diam-

eter $1\frac{1}{2}$ feet removed. Put on strychnin $1/60$, arsenic $1/30$, and thyroid 1 grain three times daily, July 18, 1916; taken the treatment irregularly. Barely visible motion of pupils to light, readily so to convergence. V. each $20/40 \pm$, S + 4.0, J. 2 —, Sept. 18, 1916. Has been taking medication irregularly: V each = $20/50$, S + 4.0, J. 3 each — J 2, jointly. Pupils dilate slowly in dark room but not to quick exposure. Patient could not be induced to return any more, but showed demonstrable improvement in the four months of observation.

The two next cases are introduced to support the thyroid and hypophyseal implication in the origin of the disease, being sisters in a family of five children, one boy of whom died at about 18 years of age, having the same degree of night-blindness and overgrowth of body in general as the sisters reported. The other two children were each seen and had normal eyes and bodies. The mother has a large goiter, which was present during all her child-bearing life, and likely deficient in providing its proper amount of internal secretion during the gestation of these children. The skeletal disproportion, and extreme adiposity, with abundance of hair on head, fit fairly the descriptions given by Cushing of dyspituitarism.

CASE 7.—L. C., aged 14, July 29, 1905. Alternating divergent strabismus as far as patient can remember. Absolutely blind when darkness approaches. Enormous weight. V each eye = $18/120 +$, J. 7 poorly, wearing S + 2.0. Visual fields much contracted; pupils normal. Cyl. + 2.0, ax. 90 each called for; does not raise vision, but dulls badly by reversal of axis. Both eyes disseminated spiders of black pigment, with splotches on retina, up to region of macula, typical retinitis pigmentosa. Gave cylinders and put on long course of small doses of arsenic and nitroglycerin; the first because of its supposed effect on metabolism, and the latter that it might theoretically cause increased waves of arterial blood supply for a short while after each taking. Next seen, Aug. 14, 1911. Fields and appearance no worse. Has seven year old sister weighing 100 pounds with night-blindness. Renewed arsenic and trinitrin. Patient's mother had goiter since 18 years of age. Next seen, Nov. 11, 1916. Has been having severe scalp trouble with bald spot on head. Walks like semiblind person. Pupils active. V = $20/100 +$ each, J. 3 readily. O. disc very white, retinal vessels small. Less pigment, especially near disc, than record would lead one to expect. Visual fields not over 8 inches in diameter at $1\frac{1}{2}$ feet. She took the treatment prescribed on each of the former occasions, about six months. Put her on the strychnin, arsenic and thyroid pills, Aug. 13, 1917. V. each $20/75 -$, J. 2+. Slight pigmentation in right, more in left retina. Optic nerves atrophied. From misunderstanding of instructions, left off treatment when overstimulation symptoms appeared, and did not resume. Gave 1 grain each of thyroid and pituitary.

CASE 8.—V. C., aged 12, Nov. 11, 1916. Sister of above patient. Enormous overgrowth of body, massive expressionless countenance, luxuriant, long, silky hair; night-blindness complete, even with full moon. Contracted visual fields, poor control or sense of direction of eyes, low ability to observe, keen memory; pupils small and react to light. V each 7/150, J. 3 —, S—3.0 each 20/100 +. With dilated pupils hard to get critical inspection fundus as eyes wandered in all directions but fundus was dark, with no red. Discs pale, vessels small. Put on half adult dose of arsenic, strychnin and thyroid, Aug. 13, 1917. Took only a few weeks, from misunderstanding of directions. Fundus slate color, optic discs and vessels atrophic. V. each 20/75 —, R = J. 1; L = J. 2 —. Prescribed thyroid with pituitary.

It was deemed worth while to try the thyroids on cases of atrophy other than atrophy in retinitis pigmentosa, in certain cases of congenital atrophy of children, with low vision and pallid nerves, but none have been followed up on which to report reliably, as they could not be induced to persist in treatment, or return for observation. For instance, about twelve years ago a boy had his eye ruptured, and examination at the time showed congenital atrophy of right optic nerve with $V = 20/60$ —, at which point it stood unchanged, as opportunity to examine recurred many times. In February, 1917, he felt need of visual help, and was put on the arsenic, strychnin and thyroid, but shortly after removed to New Mexico. In reply to a letter of inquiry in September, 1917, he sends clipping marked for 20 feet, which, when compared with test chart seems to about equal the 25 foot line, and print the size of J. 1 read at 10 inches, but as he was not tested by standard letters, the result is dubious. He took only a month's treatment.

Aug. 8, 1914, H. D., aged 18, came for dulness of sight and hearing. He was mentally defective to the extent that he could not be trusted to travel alone. He had rapid nystagmus in both eyes with $V = 20/75$ —, J. 3, and $20/40 \pm$, J. 2, right and left, respectively, not raised by glasses. No encouragement was offered as to helping the vision or nystagmus, but his mother was advised to put him on thyroids a good portion of the time for two years, in the expectation of improving his mentality. By request he returned, Aug. 9, 1917; general improvement in facial expression and manner was evident at a glance and corroborated by his conversation. Vision: R = $20/50$ +, J. 3 +; L = $20/40$ +, J. 1 —, and the nystagmus was so infrequent as to hardly attract notice.

The following were observed long enough to report on:

CASE 9.—S. McD., aged 64, Sept. 26, 1912. Right eye blinded by an iritis fifteen or more years ago, pupil occluded. No light perception. Left eye severely damaged eight years ago by iritis, pupil

nearly occluded, which conditions were incidentally observed several years ago when he came, in company with a brother who had an acute iritis in one eye, be being used as a text to illustrate what harm iritis could do, when not treated. Left fundus could be seen, the choroid being atrophic, but not destroyed. He had been told on a trip several years before that nothing could be done, so the purpose of this trip was to see about a deafness, which tuning fork tests showed to be in the nervous apparatus of ear. He was put on mercury with chalk, strychnin, Dover's powder and thiosinamin pills, and returned, Nov. 30, 1912. Following this, pupil was visibly contractile, $V=6/200$ with strong — Sph., J. 14—. The choroid had the color and appearance of grained oak, no red being visible, and optic nerve a mere splotch, with vessels hardly visible, and soon vanishing. It was suggested to try thyroid, and he was given 2 grain tablets three times a day. Feb. 25, 1913: V. S — $6.0 = 18/200$ —, J. 8 partly; Aug. 8, 1913, S — 6.0 , $V = 20/100$ +, S + 3.0 , J. 2 —. Dec. 1, 1916, vision about same.

CASE 10.—H. McD., aged 60, March 21, 1913. Brother to Case 9. Made one visit several years previously for acute iritis of two or more weeks' duration in one eye; other normal. Right pupil fairly large, no reflex to light. $V =$ fingers at 1 foot; only seen in outer field. General atrophy of choroid, red lining being substituted by fibrous looking tissue with symmetrical splotches containing black spots, comparable to "dog tracks," and some tallowy flakes. Left pupil sluggish to light, Vision fields full, fingers at 6 feet. Choroid atrophied, with red bands in it. Pigment massing small. Pulse 70, blood pressure 205. Put on thyroid tablets, 2 grains each t.i.d., subject to heart action. Seen in 1914: LV = $20/100$ —. July 8, 1916: LV = $20/75$, J. 6 with 5.0 D. sph. Field full. Splotches in left eye around macular area, worse than in right. Renewed thyroids, with strychnin, arsenic and iron. This patient was never seen again, but brother reported the pills excited his heart so that he gave them to said brother to take.

CASE 11.—H. D., aged 69, May 17, 1916. V. each $20/75$, S + 4, J. 4 each. R. pupil enlarged and sluggish except to reflex action. Disc atrophic. R. field contracted, tension up each eye, blood pressure 136. Gave strychnin, arsenic and thyroids. June 19, 1916: RV = $20/50$, J. 3; LV = $20/75$ —, J. 4 +. Aug. 18, 1916: Pupils both eyes active, RV = $20/40$ —, S + 4.0 , J. 2; LV = $20/75$ +, J. 6. Fatigues quickly. Oct. 18, 1916: Pupils small and active to light, V. as before. Dec. 19, 1916: V. as before, with some J. 1 —. Pupils small with normal reactions. April 20, 1917: RV = $20/40$, S + 4.0 , J. 2 +; LV = $20/75$, J. 6 —. July 1, 1917: Pupils normal size. R. immobile, but slowly dilates in dark, L. active; V. same as previous test. It will be observed the right greatly improved, and the left ceased to get worse. Oct. 23, 1917: LV = $20/75$. S + 4.0 , J. 3 +. RVS — $0.50 = 20/40$, J. 2.

This patient, as also Case 9, was quite dull of hearing, and tuning fork tests showed nerve impairment. Improvement in both cases in the hearing was marked, it requiring no more effort to converse at 10 feet on the later visits than it did for 3 or 4 at the beginning. It looks reasonable that whatever would improve the tone of the optic nerve, would do the same for the auditory. This has been borne out by the result of thyroid treatment in several cases of congenital sluggishness of hearing in children, but not enough to put them in the deaf-mute class. It has also been tried in one case of essential anosmia, in which the sense of smell was greatly increased.

It has always been an unexplained mystery as to why the particular form of optic atrophy manifesting night-blindness should be accompanied by pigmentation of the retina, and likewise why all cases of night-blindness do not have the pigmentation—the “Retinitis Pigmentosa, sine Pigmentum.” In my belief the disease is due to pluriglandular deficiency of the ductless glands, which may be impaired in varying degrees respectively, and in some instances either the thyroid, adrenals or the pituitary continue to functionate sufficiently. Breaking down of the adrenals would explain the pigmentation on the same basis as it explains the dark skin in Addison’s disease. The part played by each of the ductless glands individually cannot be exactly gauged in the present state of ignorance of the ultimate physiology and pathology of said endocrine glands.

It is well known that mild stimulation of any organ or function, followed by due rest, is strengthening of said organ; but excessive and long continued overstimulation is followed by exhaustion either fatal to recovery or making restoration long delayed. In this connection the great experiences of the world war are to be considered, which to me are the strongest evidence of the dependence of night-blindness on the ductless glands.

French, German, and later English statistics show great numbers of night-blindness in the trenches, as compared to same in times of peace; these reports also come from Asiatics in British service in Egypt. *The Journal of the American Medical Association*, Sept. 9, 1916, has the following abstract:

“The frequency of hemeralopia extremely low in times of peace (1 in 12,000 in France, according to Walter) undergoes an augmentation in war time. At Verdun, Dr. Bordier found 8.78 per cent. in ocular examinations during the winter of 1914-1915. At the same period there occurred in the German army a grave epidemic of this condition. More recently Vegers has observed it in the proportion of 10.2 per cent. This high figure depends on many causes, some peculiar to the present war

which presents conditions different from those which prevailed in the past, such, for example, as the higher mean age of the combatants. The hemeralopia in this war differs from that which has been described in previous wars in two respects: In the first place, there has been no epidemic, and the revictualing of the army being satisfactory, there have been no crises of hemeralopia due to privation and physiologic misery. By reason of selection, symptomatic hemeralopia is rarely found more in military than in civil life. In most cases it seems related to some vice of refraction, and principally to myopia.

"The condition has been observed among volunteers in the early months of the war, among officers of the regular army, and army officers, and because of these facts and others contained in the report of the commanding officers and because of the isolated character of each case, it is evident that, in the presence of war, hemeralopia is a symptom rarely invoked by malingerers.

"Except to those cases traceable to general disease (of liver and kidneys) or to ocular fatigue or disease, there seems to be no satisfactory treatment for the condition.

"With regard to military employment of patients suffering with hemeralopia, * * * * * it is necessary to point out that the condition appears to be scarcely, if at all, curable * * * * *."

This quotation is made for the value of its statistics; also, to show that those having this disease were subject to long continued stimulation of watchfulness, loss of sleep, or emotions accompanying the life or death struggle, which would at first increase the endocrine secretions, and later exhaust them. Theoretically, at first there would be an oversharpeness of night vision followed later by its collapse. In some preliminary remarks on this subject before this Society last year, the essayist ventured the prediction that, with the rest following peace, these cases would not develop retinitis pigmentosa beyond the normal low rate percentage, but were only functional, and would return to normal. This is borne out by a statement in the *Lancet*, in June, 1917, which says that most cases of hemeralopia in soldiers pass off with rest and relief from duty.

The British Journal of Ophthalmology for Aug., 1917, also states night-blindness is common in British and Indian soliders. but no organic disease is visible. Weekers, in *Archives of Ophthalmology*, March-April, 1916, reports night-blindness in 10 per cent. of examined cases; refraction errors frequent. He attributes it to nerve shock.

Landolt (Paris Ophth. Soc.) reports much night-blindness associated with uncorrected ametropia, which he treats by rest, rich diet, strychnin, arsenic, cod-liver oil, iron, potassium iodid, etc.

Eason in the *British Journal of Ophthalmology*, August, 1917, reports much night-blindness in Cairo and Alexandria, due to simulation, malnutrition, etc., a few real retinitis pigmentosa.

As pointing to the specific disturbance of the adrenals from these war conditions, the following is valuable: *The Journal of the American Medical Association*, June 16, 1917, in a review from *Russkiy Vrach*, Petrograd:

"Yushtchenko relates that he has encountered a number of cases in soldiers which would have been classed in with traumatic neuroses if it were not for the fact that the men presented certain symptoms which we are accustomed to encounter with Addison's disease, especially the pronounced bronzing. Analysis of the cases shows further a deficiency in the functioning of both parts of the suprarenals."

Ophthalmic Literature gives the following title "Fusita—Relation between Adrenals and Retina. Nippon Gank Zasshi—September, 1916." An effort was made through the courtesy of a friend in the Army Medical School at Washington, to ascertain the substance of this paper, but the journal was printed in Japanese-Chinese characters, and several Japs turned down the task to translate it.

Another reference of value is from *Progrès Médicale*, Paris, March 24, 1917, 12 p. 95, also from abstract in *The Journal of the American Medical Association*,

"Thyroid-Parathyroid Treatment of War Neuroses. Blanc comments on the remarkable frequency of abnormal thyroid functioning in the men on active service. He recalls that the thyroid is peculiarly susceptible to emotional stress, overfatigue, and defective hygiene, which readily explains why so many of the troops present evidences of abnormal thyroid and parathyroid functioning. He has found the oculocardiac reflex a good index of conditions. With these functional disturbances of the nervous system this reflex is usually exaggerated, indicating vagotony. Under thyroid or parathyroid treatment, or both, this reflex disappears, or becomes inverted as recovery progresses."

The first published reference which came to my notice of giving extracts of the ductless glands in the eye disease, was by Dr. S. B. Muncaster, of Washington, in the *Ophthalmic Record* of July, 1912, in which he advocates a tablet of 1 grain thyroid, 1/60 grain adrenals and 1/200 grain sodium cacodylate in retinitis pigmentosa and choroiditis when there are old pigment deposits, at the beneficial effects of which he expresses surprise. Of late some remarkable results are being reported of pituitary body disease treated by glandular extracts, either by

mouth or by injection of pituitrin. In the *Archives of Ophthalmology* for March, 1917, are two very elaborate articles on the hypophyseal relation to retinal disease with treatment by its glandular extract, narrating improvements that would hardly seem credible if emanating from obscure sources. One is by Drs. DeSchweinitz and How in Philadelphia; the other by Drs. Elsberg and Krug in New York. The latter give a reference to the Proceedings of the Royal Society of Medicine, 1915, Vol. 8, No. 3, sect. on ophthal., p. 32, in which Eason described a patient who was practically blind, who recovered normal vision and nearly a full field in one eye after thyroid feeding. The improvement had persisted for nine years; the patient had to take small doses of thyroid from time to time, otherwise vision would begin to fail, and headaches return.

While in New York last June, I had the opportunity to read Eason's article, which was short, with no theory or explanation as to why thyroid was given, or how it was supposed to act, but as his case was treated in 1907, it is probably the first on record in which this treatment was applied. Let us hope this will not prove a bubble—fair to look upon, but ending in nothing.

THE INTERNAL SECRETORY SYSTEM AND THE EYE

ANOTHER YEAR'S EXPERIENCE

ROBERT SCOTT LAMB, M.D.

WASHINGTON, D. C. *

To those members of the Academy who have listened to my papers on this subject, or have read them, I bring additional matter for consideration. My interest is lively and my practical use of organotherapy is daily and constantly increasing in the number of diseased conditions from various causes which prove to be amenable to such means of treatment. One does not care to put oneself in the position of being too hasty in drawing conclusions as to the specific efficacy of any preparation—more especially those new ones whose nature and source are not beyond the shadow of doubt as to accuracy and exactness. Therefore, I have waited before going much into detail until several years' use has demonstrated to my patients, my medical brethren, and myself, that satisfactory results are obtained with these substances, added to the usual local methods of treatment, which were not probable of attainment by methods in use prior to the introduction of animal substances in therapeutics.

I have already stated that to get the most valuable and intelligent viewpoint of this therapy one must refresh his memory by a review of the embryology, morphology, anatomy and histology; and in addition the constant review of the experimental physiology of the endocrine system. There are many who cannot spare the time from a busy life to take the matter up in any such complete fashion; still they should know what they can of the results of the efforts of others who have studied, as indicated above, and applied this therapy in the diseased conditions of the eyes.

It is to such, especially, that what I have to say may bring useful suggestion, in order that many obstinate, intractable cases may, with the additional help of organotherapy, go on to relative rapid and satisfactory recovery, or may be taken hold of with renewed interest and vigor.

Every one cannot take the time for analysis which is necessary to ascertain what has been the probable past course of the

relative activities of the endocrine organs or ductless glands and what influence they have had on the physical development and on the status praesens of each individual patient. Yet such an analysis must be approximated before the experimental therapy can take on the semblance of scientific investigation.

RESULTS TO BE EXPECTED

When deficiency in the secretion of any organ is repeatedly found associated with certain symptoms, and the administration of the required animal substance is begun, the coincident improvement in the condition and final disappearance of the symptoms is gratifying and satisfying—if not absolutely scientific. Nevertheless, practically and clinically, one feels justified in telling his friends in order that we may multiply the opportunities for trial of the substance in these conditions in which the aforesaid symptoms occur and so bring the matter to a reasonably justifiable basis of fact, which will be accepted by the majority of us, whereupon the use of the substance will be generally sanctioned for the treatment of such symptoms.

In view of the fact that it is my desire to stimulate a lively interest in the practical application of organotherapeutic measures, and realizing that very great elaboration of the subject by tabulating so many cases treated with a certain substance, and the results obtained, does not in any way bring a fair conception of the complicating conditions that were encountered in connection with any diseased condition treated, I have thought it expedient to write the experiences in a more informal fashion. Perhaps the experience may then contain a human element that will make them that much more real, and I hope thereby a great longing to use these animal substances may take hold of you.

The cases reported and the conditions encountered in them will not be taken in chronological order, but a few scattered here and there will, perhaps, suffice as examples of the kind of work done to illustrate the possibilities of organotherapy in ocular diseases.

Referring for a moment to my paper of last year, I have been fortunate in having two more cases of acute inflammatory glaucoma which have been controlled by the use of pilocarpin and adrenalin by hypodermic injections; and subsequently a trephining in one case and iridectomy for glaucoma in the other were successfully performed with good visual results.

FIRST GROUP OF CASES DUE TO EPITHELIAL LETHARGY AND
TARDY REGENERATION.

One of the most common occurrences in our clinical experience and private practice is to see a case of blepharitis marginalis associated with phlyctenular ophthalmia; for many years it has been known that such cases do better without too much starchy food. I remember Ramsay some years ago in his discourse to us on phlyctenular keratitis especially emphasized the importance of curtailing the use of sugars and starchy substances. I am not so sure as to his reference regarding starches, but it follows, logically, they should be included. Disregarding other factors such as the shape of the face, the condition of the hair, and the bony growth of the individual, which might lead us to the probable dysfunction of the anterior pituitary, we have learned by experience that very close association between apparent sugar tolerance and hypersecretion of the anterior pituitary substance exists. * * * I say apparent sugar tolerance because since Cushing¹ made his statements regarding sugar tolerance in his book, Wilder and Sansum² have shown by recorded experiment that

"In none of the cases of hypophysis disease has there been any increase of the glucose tolerance above the normal limits, but on the contrary, a tendency toward decrease, which would seem to imply that increases of glucose tolerance in such cases, following alimentary administration of glucose, are due to delays in absorption rate rather than to changes in the intermediate metabolism."

It would therefore seem that Cushing had no conclusive evidence on which to base his statements regarding sugar tolerance.

An explanation has occurred to me that perhaps an apparent sugar tolerance was the result of an increased peristalsis due to pituitary secretion; which prevented the constipation usually incident to the ingestion of large amounts of starchy food. The intestinal toxin absorption occurring from fermenting starches could easily be responsible for malassimilation, malnutrition and lowered tissue resistance; all of which might easily be improved by the peristalsis resulting from the pituitary secretion.

Effect on Epithelial Growth.—We know also that anterior pituitary substance, as in the case of tethalin, has been proven to be especially potent for the acceleration of the epithelial

1. Cushing, Harvey: *The Pituitary Body and Its Disorders*, Philadelphia, J. B. Lippincott Co.
2. Wilder and Sansum: *Arch. Int. Med.* 1917, **19**, 311-334.

growth. Realizing then that the tissues with which we are dealing in this disease may have an epiblastic or ectodermic origin, we apply our usual local remedies, such as yellow oxid of mercury—and if there be ulceration—a little atropin, calomel powder, hot applications or other means, even including, as is our custom in many cases, the use of external canthotomy; in addition, we give internally anterior pituitary substance. It is a pleasant surprise to see the rapidity with which such cases resolve themselves into a cure.

Another application of the usefulness of anterior pituitary is shown by a patient—a stonemason—whose eye was lost through a flying “spall,” puncturing and perforating both anterior and posterior walls of the eyeball and destroying the lens and vitreous. The eye was removed and a lead-free glass ball implanted, the stitches cut out and the glass ball showed a tendency toward extrusion. It was the first case of the kind that had occurred in the last twelve years of my practice, in which following enucleation, I have almost invariably implanted a lead-free glass or gold ball. On inquiry, I learned that two operations had been done in the past six months—one for appendicitis—so his general condition was undoubtedly thoroughly well run down and he was quite weak. He returned to the hospital and the one glass ball was removed and a second ball, a few millimeters smaller, was introduced. Before the second implantation, at which time the first ball was removed by me, I gave him anterior pituitary substance to use for one week. He continued its use throughout the period of operation and recovery and there was no sign of the tissue giving way; furthermore, he has a perfectly smooth and beautiful conjunctival covering over the stump. Coincidentally the man’s general health improved, he gained strength, his appetite increased, and altogether his general physical condition is much better.

Another case comes to mind—cancer of the nasal third of the left lower lid at which the patient had been picking for a year before coming to the office. That very day in the office under cocain and adrenalin the growth was removed, sent to the laboratory and a diagnosis of epithelioma was forthcoming from the pathologist. The growth was about the size of a small hickory nut, and of course the hole left by the cautery was slightly larger—from 15 to 18 mm. in diameter and about 12 mm. deep. The local application following the removal was bichlorid ointment; magnesium sulphate—saturated solution—was used for hot applications for the reduction of the swelling following

the cauterization. Internally, she was given anterior pituitary substance. It is with difficulty today that one can tell which lid has been operated on.

Whereas some of these recoveries that I have had may seem incredible and miraculous, I am inclined to ask your indulgence until the matter is sufficiently proved to enable us to determine just how much credit we can give to the particular substance mentioned, and how much might be due to other beneficent influence. As for myself I am satisfied these substances have had a very decided influence on the metabolic processes within the tissues.

GROUP OF CASES WITH ENTIRELY DIFFERENT CHARACTERISTICS

The nonexophthalmic type of hyperthyroidism without and with asthenia is presumably due to inactive chromaffin tissue. These patients have chorioretinitis, widely dilated pupils, slightly increased intraocular tension, overacting heart, flushed face, headache, beginning to be incapacitated for their work and are bordering on the so-called neurasthenic or nervous breakdown state. In addition to local remedies used, such as mercury friction to temples, pilocarpin and dionin, and hot applications, patients have recovered speedily and satisfactorily and have been free from headache, being assisted in this by the administration of posterior pituitary and gonadal substance—either ovarian or orchic—with the addition of suprarenalin substance in those patients complaining severely of weakness which they experience in the course of their daily work.

A patient whose father died of exophthalmic goiter had absorption of retinal pigment and increased intraocular tension, so severe as to be quite alarming. After removal of the tonsils together with local medication and the administration of lutein and whole gland pituitary, the patient has been enabled to overcome a condition of invalidism and is now leading a very strenuous life, being secretary to one of the busy men of the Northwest section of the country and is still having energy enough left for her social engagements.

Some may take issue as to the efficacy of the internal medication in this case, because the tonsils were removed and were a source of toxin affecting the entire system. I wish to point out that if this patient attempts to go without these animal substances there is an immediate slump in her physical well-being. I would not, however, for the world be understood as thinking that the removal of the tonsils did not do good; as a

matter of fact, I do not think any permanent results could have been secured for this young woman without the removal of the tonsils.

Some of our members may be so located that they may have to assume the entire responsibility for this kind of medication; others may possibly be able to obtain the services of a good diagnostician and perhaps leave the question of internal medication to him. My purpose is merely to call attention to the beneficent influence of these substances when administered in these cases after a correct diagnosis of the insufficiencies has been made.

GROUP OF CASES WITH LOWERED METABOLISM FROM CONGENITAL OR ACQUIRED ENDOCRINE INSUFFICIENCY

(1) *Hypothyroid Conditions*.—Those cases of a myxedematous type in patients 45 years of age who come to get relief from swollen, baggy lids, respond very quickly to thyroid medication.

(2) *Those Patients Whose Condition Is Due to Vasomotor Exhaustion*.—Under this group one case of peculiar interest is as follows:

REPORT OF CASE

A young man connected with one of our scientific institutions has been in the habit of doing much work by strong reflected illumination on specimens which he observes through a binocular. He came to me with a very marked chorioretinitis with exudates, complaining of light and such fatigue as to be unable to walk a block without resting. He was 32 years of age. Careful analysis indicated that during his entire life he had been deficient in posterior pituitary substance and that because of much intensive work he had so drawn on his suprarenalin glands that they were beginning to disintegrate. The skin on his forehead was about five-eighths inch thick, and that of the entire face was loose, thick and greasy. He had been for several years affected with a so-called psoriasis, for which he had taken a great deal of treatment. The dandruff in his hair was from an eighth to three-sixteenths inch thick—his elbows, knees and various parts of his body were similarly affected. He could no longer stand the light which it was necessary to use to outline the detail of the specimens on which he was working; he had certain sketches to complete before a special bulletin could be published. You can easily imagine the despair that this man felt when he realized that each day he was becoming hopelessly weaker and incapacitated. In addition to the local remedies such as pilocarpin and dionin, mercury friction to temples, hot applications and a protecting eyeglass, he was given posterior pituitary, orchic substance and suprarenalin substance, one tablet of each three times a day.

It was very gratifying to see this young man improve in every way—in the course of several weeks walking 5 or 6 miles every day to and from his work and feeling like a fighting cock. An interesting feature of his case was that after six months of improvement and health a complication arose in his domestic affairs; his wife was due to have an accouchement and had coincidently some of the nervous manifestations that occasionally occur with women of a nervous temperament. He was so tremendously upset that he came in feeling almost as desperate as when I first saw him; fortunately there was no coincidental chorioretinal inflammation, and I told him to cheer up and double the dose, that is, taking two tablets of each substance, and admonished him as he felt better and the crisis passed he could fall back on his previous dose which had been sufficient to carry him along for so many months in comfort and relative good health. A week later I saw him and he remarked: "Those tablets are marvelous," and said he was feeling just as well again within twelve hours of the increase in the dosage, as he had before the period of depression.

Another case under this group was due to excessive indulgence in sexual practices.

This patient was referred to me after he had been the rounds; for ten years he had been suffering with migraine; he had been to a number of oculists, among whom were some of the best in the country. He had with him two pockets full of eyeglass cases—the contents representing the prescriptions of these men. He had had some eminent advice and had consulted gastro-enterologists, roentgenologists, surgeons, etc., and was a typical hypochondriac. On inquiry as to his habits I learned he had an exceptional libido. For instance, no matter how often—several times a day—he might return to his home during business hours (being an outside man in a real estate office and so finding it convenient to drop by the house) he would feel impelled to have intercourse with his wife. In fact, in meeting women folk whom he knew, or sitting around the card table socially in the evening, he would sometimes have an orgasm. The result was, of course, a depletion of all his vital forces. The migraine was controlled within the first two weeks by the use of protecting lenses, carefully ground, and local treatment such as pilocarpin and dionin, and mercury friction to the temples. Knowing, however, that it would be impossible to get continued control over the migraine, and that there would be recurrences, as there had always been in the past ten years, I secured the cooperation of the diagnostician on whom I chiefly depend for a diagnosis of disturbed conditions of the general system. Because of a knowledge that certain experiments on rats had proven the possibility of causing testicular atrophy, the patient was confronted with the information that it would be absolutely necessary to attempt to cause a lack of desire on his part for sexual intercourse, and his cooperation was obtained; he being willing to do anything to get better. It seems that his income had dwindled from \$10,000 a year to a

haphazard sum of from \$50 to \$100 a month. He was weak and worn out and had the tendency the same as so many of these patients to weep on the slightest provocation. He said if I could not help him he would take the suicide route out of it all. He was put on a dosage of posterior pituitary, lutein and supra-renalin. When seen about a year afterwards he told me he was continuing to use the tablets (which he was educated to use intelligently for the first month or six weeks) and he had forgotten entirely how his migraine headaches had previously felt—being entirely free from them. Of course, the man's physical condition can never be perfectly normal. He goes as much as two weeks and longer without any desire for intercourse and feels well beyond his fondest hopes, is able to attend to business in a more rational manner, and is really succeeding in reestablishing himself in the business world as a reliable individual.

There is nothing new in the idea that too frequent intercourse is deleterious to the eyes. Quoting from *The Ophthalmic Record*, Volume 24, page 453, in an article by Dr. Lewis H. Taylor on a little book he found by Dr. Walter Bayley (1529-1592):

"A modest use of Venus performed in the feare of God in due time, when the meate in the stomacke is digested, and nature desirous to be disburdened, is to be allowed; so immoderate and unseasonable use thereof, doeth of all things most hurt the sight, and soonest induce blindness."

It would be an imposition to continue to recite the histories of other cases; it is sufficient it would seem to me that the foregoing cases are neither the mildest nor yet the worst, even though the last case seems bad enough. I have among my cases one of a similar character, only very much worse. However, I will not include it in this recitation.

In addition to these, numerous cases of neuritis associated with headache, eyeache and migraine have come under observation and control.

Perhaps, after what I have been able to bring before you today you will pardon me for returning to you again this year with the title of my paper practically the same as last year's paper. Again it would seem from the foregoing that it was absolutely necessary and imperative that some one should come forward insisting that advantage should be taken of the many opportunities which each and every one who does ophthalmologic work has to look more closely into the environment and previous history of his patients, and by so doing put himself in the possession of knowledge that will enable him to be of the greatest assistance to his patients; and to put our avocation where it will not only be of a greater economic value to the community.

but redound to itself such approval of its methods and results, that fakirs and charlatans will have little chance to get any foothold.

In conclusion, I may say that with the limited time at my disposal it has been impossible to do more than bring you to the top of the hill of observation from which you can look off into the vast, fertile fields of the possibilities for the growth of endocrine medication in ophthalmology, and there leave you to ponder.

DISCUSSION

DR. S. MUNCASTER, Washington, D. C.: Drs. Jones and Lamb both have proved that the ductless gland secretions are helpful in these cases. In 1912, in the *Ophthalmic Record*, I quoted some experiences with patients in which retinitis pigmentosa and old choroidal trouble were present. At that time I prescribed thyroid extract, 1 grain; adrenalin, 1/30 grain, and sodium cacodylate, 1/200 grain. Since that time the formula has been changed to a shotgun prescription of a number of ductless glands. This retinitis pigmentosa patient I have kept under observation. She is a school teacher, and at the time that I read that article a slight improvement had occurred in the amount of pigment deposit. I advised her at that time that she had better give up school teaching, but she would not do so and has been teaching six years, but her eyesight is better than when she first came to me. I do not claim the deposits have cleared up, but the inflammatory diseased tissue has cleared and afforded her a better field of vision.

I also cited the case of a cretin who had glaucoma and beginning cataract, a very stupid looking child. I kept her under this treatment for six years. I saw her about a month ago, and her uncle, who is a physician, says she is the most intelligent member of a family of five, though she was at first like a stupid idiot.

I think there is no doubt that these substances have come to stay. Now we must treat eyes with these ductless glands. The reason we do not get good results is because they are not made fresh. The only way to do is to have the druggist in your town have a substance ordered direct from some chemist you can rely on. You can only use it for two years; after that you will get no results. A great many druggists are apt to have them on the shelf for five or ten years and use such products for prescriptions, and they will do no good. I think the substance, to give good results, should be not over three or four months old. I prescribe a tablet of late called Tablet Thyrodyl No. 1. It contains thyroid substance 1 grain, adrenal substance 1/60 grain, pituitary body 1/30 grain, pancreatin 1/8 grain, sodium cacodylate 1/200 grain, saccharum lactis (Merck) 1 grain, tragacanth 1/8 grain. It is compounded by the Perron Laboratory, Newark, New Jersey. This tablet is taken after meals three times daily.

DR. R. S. LAMB, Washington, D. C.: Regarding the idea of syphilitic infection, chiefly of a congenital character, there is nothing that disintegrates the internal secretory glands like acute infections, and among these probably none is more industrious and virulent than is the syphilitic virus. Many of these congenital syphilitics have definite malformations. Their bodies, their heads, their legs are all deformed, as you all know; and I believe that the *modus operandi* of the development

of retinitis pigmentosa is from syphilis disintegrating the endocrine system.

Dr. G. B. JOHNSON, Franklin, Pa.: Drs. Jones and Lamb are deserving of the congratulations of the eye section of this society for their lucid and timely papers on the subject of organotherapy, as the vast majority of ophthalmologists have not given the subject the attention which it deserves in therapeutics of the eye.

When used intelligently, hormone therapy is of inestimable value in the treatment of diseases dependent on derangement of the internal secretory organs, but when wrongly used, dire results may accrue.

This is exemplified in the case of three cretins who were treated with thyroid extract at the Pennsylvania State Institution for Feeble Minded at Polk, with marked physical and mental improvement. On the contrary, a case of obesity, which previously was in good health, developed acute diabetes shortly after commencing the use of thyroid extract for the reduction of fat.

The tendency in the past has been to administer too large doses of thyroid preparations. Very small doses in many cases produce the desired result. The initial dose should not be over $\frac{1}{2}$ grain, which may be increased to $1\frac{1}{2}$ grains, given three or four times a day, depending on circumstances. Even $1/20$ grain at a dose, given over a long period has been very beneficial in cases.

Adrenalin which has been mentioned, is another exceedingly valuable organotherapeutic agent, when properly used; but great care must be exercised in the administration of this potent remedy in cases of adrenal overactivity, such as occur in diabetes mellitus, hypertension, etc. Weak adrenalin solution instilled into the conjunctival sac, has caused serious palpitation, general weakness and dyspnea, according to Harrower; which leads me to believe, that untoward results sometimes attributed to cocain, are in reality due to the adrenalin, which is so frequently used in conjunction with cocain.

One of the most valuable of the organotherapeutic agents is parathyroid extract; insufficiency of which, according to Schiotz, may lead to cataract. Berkeley of New York has cured many cases of paralysis agitans by the use of this agent. And I have used parathyroid extract with very beneficial results in a case of paralysis agitans in a member of my family. The agent should be used hypodermically in dosage containing $1/50$ grain of the extract, and continued over a long period of time. The preparation used, was an acetic extract of the fresh glands, as recommended by Dr. Berkeley.

Dr. H. M. LOKEY, Atlanta, Ga.: I am not going to say anything in regard to the therapy of the thyroid gland, but Dr. Jones came from my part of the country in early days, and I wish to record the difficult diagnosis of retinal or choroidal cases in the negro race. In studying the fundus of the colored population, instead of the ordinary pink, we frequently have pigmentation that in a white person you would call pigmentosa, but in the negro it is normal. In quite a number of years of work among them, I very rarely saw a retinitis pigmentosa or choroiditis, but frequently in syphilis, and nearly all are infected, we see iritis or irido-cyclitis. We do not get so many retinal inflammations as among the whites, but in differentiating in the colored race between the normal and abnormal fundus one becomes accustomed to look for the pigmented area, and if not careful you will make a mistake between the normal fundus of the colored population and retinitis.

MAJOR GREENWOOD: I want to crave your indulgence in discussing this paper on the question of retinitis pigmentosa as applied to the Army. Last week I was asked in the Surgeon-General's office to make a list

of eye diseases for the new manual. These diseases are numbered and the number used in the army to designate the disease or injury. I have changed some old terminology and for night blindness I have used the term "nocturnal amblyopia." In the examination of recruits the fields are not taken so some of these men having retinitis pigmentosa, with vision up to the requirements in each eye and a very contracted field, may be accepted. Such are a distinct danger in the Army as they have to be led around at night and are typical of the congenital form, but some develop nocturnal amblyopia as a result of their life in the trenches, giving a temporary condition. Those who examine troops must be on the lookout for this condition. Such men can easily get into the Army and then become a menace to their associates on guard duty.

DR. H. R. JOHNSON, Fairmont, W. Va.: I would ask Dr. Lamb to tell what are his special indications for the administration, the anterior, or the posterior, or the whole body of the pituitary.

DR. F. PARK LEWIS, Buffalo, N. Y.: The fact that five papers out of the sixteen on ophthalmic subjects read at this meeting have to do with the relation of the ductless glands to eye conditions emphasizes the fact that we are not mechanicians merely, but that the ophthalmologist must take into account the entire human economy and especially its physiology in considering the eyes. As time passes we are apt to forget to whom we are indebted for our first impressions concerning important subjects. It is recalled to me today that it was Dr. Risley who years ago urged on us the importance of the relationship of the ductless glands to various eye diseases. I had quite forgotten that this was so, until now that we are all becoming more interested in the subject I recall with what patience and foresight he directed our attention to this subject many years ago. We are indebted to Drs. Jones and Lamb for further enforcing the value of this subject on the Academy. We are now only on the edge of what we shall know as to the relation of these glands to various eye and ear conditions, and even yet we are unable to definitely and clearly formulate the activity of each gland in distinction from the others. The prevalent idea that the thyroid substance should be given only for the reduction of obesity in elderly people is misleading. It is quite as useful in the opposite condition; that of the young who are prematurely aged, in whom the skin is dry and harsh and in whom the hair turns gray too early. If we could definitely formulate the principal activity of all the glands it would greatly aid us in their discriminating use.

DR. CLARK W. HAWLEY, Chicago: This is a subject in which I am very much interested indeed. Dr. Lewis has taken out of my mouth that which I most wanted to say, namely, to give Dr. Risley the benefit of having first called our attention to the fact that many of the uveal tract diseases that have seemed indefinite, were caused by focal infections of the general system. Entirely independent of Dr. Risley, through my own misfortune five years ago of having chronic cyclitis I took up the study of this. I do not understand why I did not know he had done so. I knew there was no specific reason for it and through an accident I was almost absolutely cured; I took up the study of autointoxication from the lower bowel and in the past five years I have made a good many discoveries along that line, so that two years ago I published a paper on autointoxication and its relation to uveal disease. In this paper I dilated on the relation of glaucoma to autointoxication from the lower bowel and some of my results are very remarkable. Today I have been delighted with this paper. In my study I am going to add to my work along that line and add to the benefits already derived by taking care of the lower bowel. Some who study the pituitary commence

at the head and work down, but I commenced at the wrong end. Perhaps we shall eventually meet.

DR. JESSE S. WYLER, Cincinnati: I believe that there is no such thing as an increase in pigment in this form of retinitis, but it is really an agglutination along the blood vessels of the anterior layers of the retina of some of the previously existing pigment. This has been demonstrated experimentally by both Hess and Roemer who have been able to agglutinate the entire pigment layer. My experience with thyroid has been most unsatisfactory in three cases. The one which stands out most clearly in memory is that of a trained nurse, with absolutely no specific origin to her disease. I have used thyroid extract, in her case, in doses of from 3 to 5 grains, three times daily over long periods. Her visual fields are becoming more tubular and her night amblyopia has increased to such a degree that she is unable to navigate alone. My other cases have been equally disappointing and in view of the many constitutional disturbances, I certainly do not favor the promiscuous administration of thyroid.

DR. S. D. RISLEY, Philadelphia: I am highly honored by your chairman's invitation to speak. I have been deeply interested in this symposium on organotherapy. My published observations to which your attention has been called so kindly by Dr. F. Parke Lewis were, it is true quite original, but I made no claim to priority. Since their publication, however, the passing years and increasing experience have served to deepen my interest and confirm the opinion then formed of the value and importance of thyroid medication in a considerable group of nutritional diseases. To this group belong many cases of corneal and uveal disease, especially interstitial or parenchymatous keratitis. I have seen a large number of these patients of late years coming from an orphan asylum, a fact which of itself implies a probability of unfit parentage; the possibility at least of a vicious start in life. A tuberculous or syphilitic parentage stands almost certainly for impaired vigor in the offspring if not for a positive or actual infection. If in such children the avascular cornea should be involved in the general impairment of nutrition it is a fair deduction to conclude that other tissues and organs would suffer in like manner, namely, the thyroid gland. Now we are aware of the very important function this gland exercises over general nutrition. It seemed to me rational, a priori, to reinforce an impaired thyroid secretion by the administration of the gland. The first trial was in a young man, aged 20, without a syphilitic history but who had been forced to labor from boyhood; that is to say, to live during the years of physiologic development under untoward conditions. He was led to the clinic blind except for light perception with parenchymatous keratitis, marked mental dulness, exhibiting many phases of a myxedema and both corneas showing a salmon colored injection. He had been persistently and well treated for a long time by the usual methods employed in interstitial keratitis without benefit. I administered 3 grains three times daily of the desiccated pulverized thyroid gland of the sheep. In a few days his mental apathy vanished; he seemed a changed creature. His eyes rapidly improved and he was discharged from the hospital after a few months with a vision of $\frac{1}{3}$ and returned to his work in apparently good health, certainly in a cheerful, hopeful mood.

The result in this case and two others together with the underlying theoretic considerations were set forth in a paper presented to the American Ophthalmologic Society and published in the transactions and also in the *Ophthalmic Record* for July, 1908.

I have not trusted the various extracts of the thyroid found in the

market but used with marked success the pulverized desiccated gland of the sheep prepared by my own druggist.

DR. E. L. JONES, Cumberland, Md. (closing discussion): In regard to what Dr. Risley has said of the fresh thyroid, I have not been able to get this, but I send every prescription to one druggist, otherwise patients take it where they please. I do this to keep his stock moving and I ask him to try to get it originally as fresh as possible.

Dr. Lamb spoke of the etiology of syphilis in these cases. I believe in using the thyroid we probably get rid of something else acting in conjunction with syphilis. As to Dr. Jobson's remarks on the danger of overdosing with thyroid, you know if you are working with a sharp tool you should watch where you are cutting. I do not claim we are reading the final chapter in this subject, but simply the opening chapter, and I hope it will be worked out in time. Of course, we will have some failures from time to time.

As to the dose of thyroid, I believe in giving according to the individual under treatment.

DR. LAMB (closing discussion): Dr. Jobson brings out something that seems to require a warning; a great many are apt to be misinformed by the trade desiring to sell a given mixture of glands. I think that of the congenital syphilitics, many die so early that they will not show it clinically.

Nocturnal amblyopia is a good term. I have three patients in one family, and there is a history of one uncle and two aunts that died, who had gone blind.

Dr. Jobson asks a question which is difficult to answer definitely; but if an external gland or structure is involved, then you use anterior pituitary, but when the pigment layers are involved, then the posterior is better; in some cases you give both and the use of the whole gland is more convenient. I think accuracy in the dosage is profitable and probably it is better to use both on the same person rather than the whole gland. The latter is not quite so active.

Dr. Lewis brings that feature to my mind, the exact dosage. I think that most essential. You can tell the results you are going to expect with a careful analysis of your case and then prescribe one dose at a time, so you know where you stand.

Dr. Hawley speaks of the conditions that we are encountering all of the time, the toxic side, which may come from any part of the body. The bowel certainly has a decided action on these glands. The toxins are bound to cause changes in the metabolic processes.

Dr. Risley brings out the defense mechanism. This is very important. The thyroid sensitizes the activity of the other glands, but the suprarenal often "*gives out*" as we go through life. The spots on the face that appear many times in Addison's diseases are important. The thyroid activates the metabolic process of the entire system. We must keep in mind the effect on the heart. Many patients who take antifat remedies become so sensitive they almost "jump out of their skin."

UNRECOGNIZED CHRONIC SIMPLE GLAUCOMA. EYE STRAIN AS A CAUSE. SUGGESTIONS FOR FIELD TAKING

EDWARD J. BROWN, M.D.

MINNEAPOLIS

According to the United States Census Report for 1910 on the causes of blindness 31.2 per cent. more women than men were blind from glaucoma. These figures have added significance when we learn further that of women 20 years old and over gainfully employed before vision was lost, seamstresses and dressmakers suffered three and one-half times as frequently as women in general.

Unfortunate personal experiences early in life convinced me of two things, namely, the very great importance of eye-strain as a cause of discomfort and disease, and the great lack of appreciation of such facts on the part of the medical profession. After I had wasted two years following graduation from college as a result of such professional incompetence, one of the most brilliant specialists of the Middle West used atropia in my eyes for an hour and gave me convex spheres which were useless. Three months later Dr. E. Williams and his associate, Dr. Ayres of Cincinnati, gave me an efficient examination and corrected my 2 diopters of hyperopic astigmatism. The work of those two men was an inspiration for me. Such men were rare among the oculists of forty-two years ago, but there were a few of them. For example, in 1895, Mrs. H., a physician, aged 54, consulted me. There was a slight concavity of both optic discs and the inferior nasal third of the left field was contracted from 10° to 20°. My taking the fields led her to remark that when she was a young woman, she had consulted Dr. Williams of Boston and that he had taken her fields very carefully. My impression is that the great majority of ophthalmologists are only aroused to the need of field taking by some marked symptom or physical sign. When I studied ophthalmology at a famous school in New York City in 1888 and 1889, there was no such thing as a perimeter or tangent plane in the hospital. Hirschberg in Berlin had a perimeter but I seldom saw it used. A patient with normal corrected vision, normal pupils, apparently normal tension and only a so-called physiologic cup of the optic disc with perhaps a slight pale-

ness and concavity of the temporal half, is seldom subjected to a perimetric examination. The result is that incipient cases of glaucoma simplex are mostly unrecognized. I have lately seen two cases of telescopic vision, both with practically normal corrected vision, and only very slight temporal concavity of optic discs, corneal anesthesia and engorged episcleral vessels to suggest glaucoma. One is a Jewish woman, aged 55, the right field mostly inside 20° , the left inside 15° , tonometer: right 22, left 25 mm. Hg. The other is an Irishman, aged 62, the right field wholly inside 15° , the left inside 10° , tonometer, 20 and 23 mm. of Hg.

In 1891, Mrs. J. M. W., aged 60, consulted me for irritable eyes, I found a mild follicular conjunctivitis, vision 15/70 and with + 1.75, 15/15. There was a physiologic cup of both discs with a slight concavity of the temporal half. Tension and fields for white were normal, but I was not satisfied and asked a man of larger experience to see the case. He smiled at my suspicions and said there was no occasion for them. Ten years later Mrs. W. returned with the same complaint. She was wearing + 1.25 but accepted only + .75 sph. which gave 20/30 vision. Tension was questionable and the fields contracted from 10° to 20° in the nasal and from 20° to 40° in the temporal half. She was advised to use eserine sulphate 1 : 6,000. Sixteen months later she accepted only +.37 cyl. axis horizontal, which still gave 20/30 vision. The pupils were 3 mm., tension apparently normal, both corneas anesthetic and the fields somewhat better than at the previous visit. She removed to a distant city and died about a year ago after some years of total blindness.

If the human eye is a product of evolution it would seem a reasonable proposition that civilization has stolen a march on evolution and made demands on the eyes of large numbers of people for which they are not well prepared. Two cases come to mind at this moment, especially favorable cases since they have only a quarter diopter of astigmatism, but there is conjunctival congestion and complaint of irritation. Both have a peasant farmer ancestry, one German, the other Scotch. Both are clerks, one in a freight auditor's office. Vision is normal and the visual fields for white taken both with the hand perimeter and the tangent plane seemed normal, but a later careful examination of the German with my triplanes shows his white as well as the color fields contracted from 10° to 30° and for white more in the nasal half. The one has slight concavity of the temporal half of each optic disc, the other a deep central physiologic cup with the temporal half of the discs nearly or quite a diopter concave. These are mere boys, aged 21 and 26, respectively.

Two other men in office work, overworked and with heavy

responsibility, head bookkeeper and chief of a department, respectively, both with only a moderate amount of compound hyperopic astigmatism and muscular errors. Both consulted me about the same time in 1904. The older man, now aged 60, had one diopter deep concavity of each temporal disc and slightly contracted fields. As vision was normal my warning was discounted and beyond necessary changes of lenses, nothing was done. After a seven year interval he lately returned with corrected vision still normal but the fields much contracted, more than 50° in the inferior temporal quadrant of the left eye, while in some meridians of both eyes there had been improvement rather than retrogression. The cupping of the discs is confined to the center and temporal half, exaggerated physiologic cups. The pupils are normal in size and reaction, corneas anesthetic, tension normal (tonometer not over 20 mm.). The other man, now aged 52, presented so little in the way of suspicious signs that I made no note of the condition of the nerve-heads in the earlier years. His refraction was cared for and his nasal sinus disease treated. A few months ago when he complained that his eyes were more or less painful, I found that his corrected vision was normal, pupils normal, both temporal discs concave, the left more than the right, both corneas less sensitive than normal, tension normal and the left temporal field contracted 20° , and both fields for color considerably contracted.

My growing interest and suspicions relative to such cases were stimulated by a paper by Professor Schoen of Leipzig published in the October, 1902, *Ophthalmic Record*. He states:

"It has been shown by microscopic examination that forty eyes which had been examined in the live subject and found to have the so-called physiological excavation, had acquired it during the lifetime. This result was confirmed by statistical research and by the fact that new born children, with a few exceptions, do not exhibit excavation. I was able to examine microscopically fifteen eyes which I had previously examined in the living subject. Besides the beginning of the excavation, I discovered anatomical changes in the ora serrata with degeneration of the ciliary muscle. The degeneration of the ciliary muscle I consider the missing link explaining glaucoma simplex."

One year after reading Schoen's paper, I had carefully noted the condition of the nerve-head in 160 cases not otherwise so diseased as to lead manifestly to field changes, and found concavity of the disc in forty-five. Of these six had one or more other signs of glaucoma aside from defective fields. Of the remaining thirty-nine only four had normal fields, and that number might

probably have been reduced had the color fields been more generally taken. The thirty-five cases had peripheral defects ranging from 10° to 30° . A paper describing this study was prepared in the fall of 1903, submitted to a very competent ophthalmologic friend in Chicago, who assured me that Schoen's views were not accepted by the profession, and finally published in the *Northwestern Lancet*, in 1905 (Vol. 25, P. 256), under the very cautious title: "Is Physiological Cupping of the Optic Disc, not Confined to the Center, Evidence of a Morbid Process?" I ventured, however, to state my tentative conclusions on this subject to the ophthalmologic section of the American Medical Association at the Atlantic City meeting, in 1904, during the discussion of the scholarly paper of the late Dr. Beard on the "Temporal Cleft of the Optic Nerve-Head."

A very limited private material afforded me comparatively small opportunity to confirm my opinions, and I confess with shame that for a time my enthusiasm was cooled by the contra-opinions of men of greater experience and wider opportunities. The experiences of such men as George T. Stevens and George M. Gould ought long since to have convinced intelligent men that contra-opinions, which are too often simply the opinions of men who either have not seen or cannot see, are of very little value. The man who does not see glaucoma till there are marked symptoms and signs with contracted fields and impaired central vision is too late to be of the greatest assistance to his patient.

Of the cases that have come to me with good vision none has gone blind or come to operation save Mrs. W. whom I have mentioned, and Albert L. who returned to me from California suffering from acute inflammatory glaucoma of two weeks duration without treatment.

The following cases appeal to me as having some significance:

REPORT OF CASES

CASE 1.—Helen D., aged 14, eyes get red on near use, slight congestion of the palpebral conjunctiva, right and left vision 20/15, under scopolamin: R. + .62 cyl. vert., L. + .25 \subset + .62 cyl. vert. Muscles: L. hyperphoria 4° , exophoria 13° , exoph. in accommodation 30° .

The eyes appeared normal, tension and pupils normal. Both temporal discs concave about 1 D., the right field contracted from 10° to 20° , the fields for red mostly within the twentieth parallel. With correction of the refraction and the vertical imbalance, and the application of a 1:12,000 solution of eserine all complaint ceased at once.

CASE 2.—Harry C. G., aged 22, peasant ancestry, price clerk,

in March, 1915, complained that his right eye had been sore for some days. A mild conjunctivitis, an infected nasal sinus, diseased adenoid and tonsils were treated. Vision good and examination for glasses refused. Fifteen months later he came with complaint of bad distant vision. R. 20/50, L. 20/70, exophoria 1° and at $13''$ 2° . A deep temporal concavity of each disc. Under scopolamin — 1.25 = —.50 cyl. hor. gave 20/15 vision in each eye. A year later the patient came with complaint of irritation of the eyes and in addition to slight palpebral congestion I found moderate corneal anesthesia. Fields for white were irregularly contracted, both nasally and temporally and in parts to 20° , and for red to 30° . Pupils and tension were apparently normal. Under 1:12,000 solution of eserine all congestion and discomfort disappeared.

CASE 3.—L. McM., aged 23, clerk in a railway office, in March, 1913, complained of having pain in the right back of head three days so he could not sleep. One year before he had been kicked in the right eye and for a time saw double when tired and on looking to the right. Right and left vision 20/15, correction under scopolamin: R. — 0.25 cyl. axis 75 nasal, L. + 0.62 cyl. axis 15 temporal. Very slight muscular imbalance. A few days rest of the eyes under the cycloplegic, and the lenses made him comfortable till near the end of 1914, when he complained of having tired eyes for a month and the old pain in the right back of the head and neck. A very slight increase of the myopia in both eyes was corrected under scopolamin with complete relief, and there was no further complaint till January, 1917, when he reported a slight headache and I found under scopolamin a reduction of the myopia in the right and its disappearance in the left, the correction now being: R. — 0.50 cyl. 80 nasal, L. + 0.25 C + 0.50 cyl. 15 temporal. Vision 20/15. Aug. 6, 1917, he returned having had such a severe pain over and behind the right ear since the day before that he could not sleep. Refraction unchanged, eyes appear normal, pupils and tension normal, optic discs, not previously noted, rather deep physiologic cups, especially the right extending to the shallow temporal margin. No anesthesia of corneas. The fields for white taken with my tri-tangent planes, $30''$ radius, and a 2 cm. object were irregularly contracted and temporally as much as 30° . For red the right field was all inside 20° and the left inside 30° . He was given one drop of a 1:12,000 solution of eserine in each eye every four hours. He slept well but had some pain the next morning, which was relieved by doubling the strength of the solution. The following morning there was again pain because of the discontinuance of the eserine. He then used the drops as ordered and after a day was able to return to the weaker solution. Has remained comfortable.

CASE 4.—Harry S., aged 21, Jewish teamster, came the last of April, 1917, complaining of sore and congested eyes. There was but slight catarrhal secretion together with some palpebral

congestion. He had worn more or less for years a correction for a moderate amount of compound hyperopic astigmatism. There was sufficient nasal and throat catarrh to account for much of the eye condition, but as the temporal halves of the optic discs were slightly concave and the corneas subnormally sensitive, I took the fields. For white the right was contracted from 5° to 10° temporally, and for red and green about 30° . The left field for white was larger than the conventional field, and nearly normal for red. Pupils and tension were normal. Under scopolamin the astigmatism was found to have been reduced from $+1.25$ to $+0.75$. In addition to the refractive correction and a boro-zinc solution he was given some weeks of conservative treatment of his nose and throat. He failed however to get relief until he began the instillation of a solution of eserin, 1:12,000, when the congestion and irritation disappeared.

CASE 5.—Mike M., aged 31, a healthy appearing Lake Superior fisherman, referred to me four years ago by Dr. Knut Hoegh of this city, for an ear and throat trouble, made no complaint of his eyes, but when he returned to me in May, 1917, for further attention, I noticed one day that the pupils were suspiciously large, 5mm., reaction normal. Vision 20/20; retinoscopic correction without dilating pupil: $+0.25$ cyl. vert., left hyperphoria $.5^{\circ}$; slight temporal concavity of both discs. Blind spots: Taken at 2 meters with 2 cm. object, R. 25×25 cm., L. 45×25 cm. and displaced down and out. The tonometer reading (McLean) was 35 mm. of Hg for each eye. The fields for white were irregularly contracted, mostly temporally, from 0 to 20° . For red the fields were contracted still more. After using eserin, 1:6,000, for two days the tonometer readings were 25 and 30 mm. There had been no discomfort and no suspicion of trouble with the eyes.

CASE 6.—Nellie O., aged 19, Finnish waitress, in 1916 came complaining of headache and I corrected her vision to 20/20 under scopolamin: R. $-0.75 \text{ C} + 3.50$ cyl. 100° , L. $+0.50 \text{ C} + 1.25$ cyl. vert. She came again in August, 1917, with complaint of headache for two weeks. Vision right and left 20/15 could not be improved. The corneas were normally sensitive, the discs unusually vascular and the slightest suggestion of a beginning temporal concavity. Careful testing with the tri-tangent planes showed general contraction of the fields for white and red, the right inferior temporal quadrant 30° and the left inferior temporal more than 40° . She was given a weak solution of eserin and has not reported since.

CASE 7.—J. C. B., aged 24, salesman, has a moderate amount of compound hyperopic astigmatism, exophoria 2° and abduction 10° . He has had much headache and while in college had a section of the right externus sufficient to correct 2° of torsion. In June, 1917, I found corrected vision normal, both corneas anesthetic, both temporal discs concave, right blind spot, 18×22 cm., left,

22x25 cm. at 2 meters, the right field for white normal except a 10° defect of the superior nasal quadrant, for red contracted 10°; the left field for white contracted irregularly from 10° to more than 50°, mostly in the inferior temporal quadrant, for red mostly inside 20° but extending temporally to 37°.

CASE 8.—The mother of this young man, now aged 50, came under my care at the age of 22. Two professors of ophthalmology, east and west, had given her concave spherical lenses, neither had corrected her astigmatism. She had 15/20 vision with — 10. and — 11. D. and quarter diopter cylinders. After two days' use of atropia the retinoscope gave: R. —9. —1.25 cyl. hor.; L.—9.75 —0.50 cyl. hor. As there were 7° of exophoria with abduction 14° the lenses were ground on 2° prisms base in. This woman has borne and reared six children, done an enormous amount of reading and writing besides much of the sewing and housework required in such a family, and has also been active in church and club life. Now at 50 her eye grounds are fairly normal save for a slight concavity of the temporal nerve-heads and her vision is better than when she came to me twenty-eight years ago. As she had been having some headache she came to me lately and I found that the axes of her astigmatism had markedly changed within a year. The present correction was accepted without complaint and gave immediate relief.

R. — 10.50 — 0.50 cyl. axis 125 ⊖ prism 3° base in.

L. — 10. — 0.50 cyl. axis 50 ⊖ prism 3° base in and 3° base down.

Reading segments: + 2.50 ⊖ prism 3° base in.

The right field for white is contracted nearly 20° both nasally and temporally, for red nearly 30° temporally; the left field for white is contracted 45° temporally and for red is mostly within 20°.

I could multiply such case reports almost indefinitely even from my limited material. Such cases seem to me to make the mystery connected with glaucoma not so much one of why a few people go blind from this form of the disease as one of why we do not all go blind with our abuse of an organ so sensitive to impressions and so frequently poorly adapted for the strain of modern civilization. Ever since the essential character of glaucoma came to be recognized the changes have been rung on drainage and secretion, on the anatomical and chemical and other physical elements in the problem, including of late the question regarding toxins and disordered secretions of the endocrine organs, but the one universal cause has been given scant recognition. The splendid work of Priestly Smith, Risley and others would seem to have laid a good foundation for at least considering Schoen's claims with an open mind.

OBSERVATIONS IN PRACTICE

What do we find in practice? Practically all the myopic eyes whose fields I have examined show marked defects. I strongly suspect that a considerable percentage of eyes as they come to us for refraction would if carefully interrogated show peripheral defects for white or colors. Nature has not yet produced the eye which can stand up under the strain of modern civilization. The small eye with a thick and tough sclerotic coat will probably measurably maintain its shape while the force of the strain to which it is subjected will tell on the ciliary region and secondarily on the nerve-head. The larger and especially the eye with a thinner and less resistant sclerotic coat will relatively yield more in the stretching of that coat, but the nerve-head in either case is apt to suffer.

CASE 9.—As I write, Charlotte J., aged 19, student, comes back after three years. She has about 5 diopters of compound myopic astigmatism, corneas 13 mm. less than normally sensitive and fields for both white and color contracted in parts 30°. The inferior and temporal half of each optic disc is concaved to the extent of about 2 diopters and there is a pronounced scleral ring. She came in only because she had broken a lens. Corrected vision 20/15.

Three cases of former patients who have come in since the above was written are perhaps worth recording.

CASE 10.—Margaret F., aged 16 months, a bright infant just beginning to walk, was brought to me, in 1909, for right convergent squint. A healthy child of intelligent Americans. After three days' use of atropia the retinoscope showed: right and left $+0.50 + 2$ cyl. axis vert. This infant not only accepted the glasses without protest but was anxious to wear them constantly. Seven years later the refraction was: R. $+ .50 + 2$ cyl. vert. = 15/200, L. $+ 0.50 + 2.50$ cyl. vert. = 20/40. In August, 1917, at the age of 9 years, the refraction was: R. $- 0.25 + 2.25$ cyl. axis 45 = 15/200, L. $+ 0.50 + 3$ cyl. vert. = 20/40. With induction of diplopia esophoria measures 16°. Both fields are contracted especially in the nasal and temporal quadrants, the right from 20° to 30°, the left up to more than 50° temporally, thus showing the greater loss of field in the better and more used eye. The fields in this case may never have been normal. Both temporal discs are slightly concave.

CASE 11.—Augusta H., aged 22, Jewish, stenographer, came a few days ago complaining of headache. She came first five years ago with the same complaint and wearing compound concave lenses of moderate strength prescribed by an optometrist. Exophoria 3°, in accommodation 13° and abduction 11°. Under proper correction of the refractive error she has been comfort-

able till the past few months. The fact that corrected vision was only 20/30 ought to have induced further investigation but nothing was done beyond eliciting that she had chronic nasopharyngeal discharge for which she refused treatment. At the recent visit a quarter diopter addition to one of the cylinders was required; vision 20/30 and 20/50, orthophoria at 20', exoph. at 13" 12° and abduction the same. Both corneas somewhat anesthetic, pupils and tension apparently normal. The temporal quadrant of the right optic disc was concave and the temporal two-thirds of the left. Both fields were generally contracted, from 10° to 30° and more in the left. The horizontal diameter of each blind spot of Mariotte at 2 meters was 25 cm.

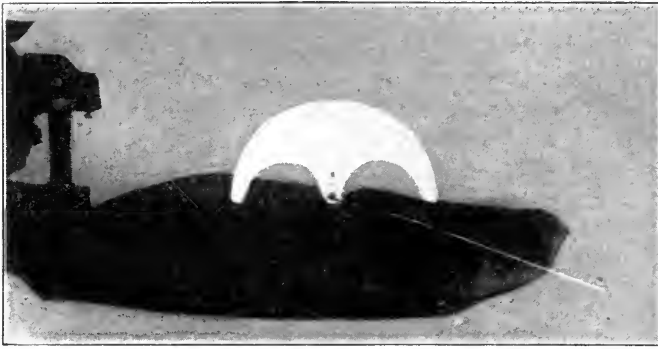
CASE 12.—Arthur P. C., restaurant owner, aged 24, consulted me six years ago for a subconjunctival hemorrhage of the right eye, the result of too forcibly blowing the nose. Vision was 20/15 and examination was declined beyond the determination of the fact of a quarter diopter of astigmatism. In August, 1917, he complained of having had five or six attacks of headache with vomiting in the morning in the past year. Pupils and tension were apparently normal but the temporal half of each disc was concave with undermined vessels, and the fields were contracted, the right from 20° to 25°, the left as much as 40° and 50° in the temporal half. Orthophoria for distance, 13° exophoria for 13" and 8° abduction. Under homatropin (followed by a strong solution of eserine till the pupils were contracted) the refraction was found to be: right and left $+ 0.50 + 0.25$ cyl. vert. = 20/15. On the second day after the use of the homatropin and eserine the eyes were entirely comfortable, the tenderness of the eyeballs having disappeared. He was given the refractive correction and a solution of eserine, 1:24,000.

CASE 11.—Mrs. K., 33, Jewish farmer's wife, came in lately complaining of pain in the eyes and phosphenes. She came five years ago saying her eyes had felt like sand for ten years and for the past eight months there had been a floating body an inch long in the right. I found nothing objective in the fundus of either eye and only noted the refraction: R. — 1. — 2.50 cyl. axis 10 = 20/40, L. — 0.25 cyl. hor. = 20/40. She returned for the completion of the examination two years later when I found: R. — 1. — 2. cyl. axis 15 = 20/30, L. — 0.25 cyl. hor. = 20/50, exophoria 6°, at 13" 16° and abduction 10°. She also had diseased adenoids and tonsils, which she refused to have treated. Now, in 1917, I find 2.5 mm. pupils which dilate but little and very slowly to several instillations of 1 per cent. cocaine, a deep temporal concavity of the right and a more shallow one of the left disc. There is a slight change of the refraction of the right eye and the corrected vision is 20/30, better than five years ago. The right field is contracted nearly 20° and the left nearly 30°.

SUGGESTIONS FOR FIELD TAKING

After using the Schweiger hand perimenter for many years

I made from a cardboard protractor nailed at a right angle to a wooden handle, the latter so fashioned that it could be placed in various meridians without touching the nose, an instrument with which the horizontal and near horizontal field could be much more easily and quickly roughly determined. A copper wire carrier a foot long attached to the instrument at the radial center, had a bend beyond the circumference of the protractor to form a handle, and was shaped at the farther end for the attachment of various white and colored test objects. Later I substituted a heavy wire handle and for the cardboard protractor a half circular sheet of copper with a radius of 6". Half circles



Practical perimeter designed by the author and constructed of a half circular sheet of copper with a heavy wire handle.

with radii of 2.5" were removed from either side to permit of necessary revolutions. This instrument is practical and fairly accurate. Greater accuracy could of course be secured by lengthening the carrier. The patient fixes his eye (or better his eyes with binocular fixation) on a light or other distant object and holds the central visual line of the instrument steadily upon the same object.

For some years I have used with more or less satisfaction a tangent plane 60" x 3" fastened to the wall at the height of 4 feet by a bolt on which it revolves. The plane is black and on the two edges in white are the degrees, on one edge for a radius of 30", on the other for a radius of 2 meters. As measurements beyond 45° on such a plane are unsatisfactory, I have lately hinged to the ends of my plane two 30 inch planes at right angles to the former, which make the instrument ideally accurate and convenient.

Since reading Dr. David W. Wells' paper on Haitz' charts, a reprint of which he kindly sent me, I have acted on his sug-

gestion to use binocular fixation in charting fields. For this purpose I have used a concave copper disc with a central one-eighth inch perforation held before the fellow eye in a long handled lens clip.

These instruments seem to me to go a long way toward meeting the indications pointed out in Dr. Peter's admirable paper of last year on the campimeter, as well as the objections brought out in the discussion.

My experience leads me to the belief that an examination of the eyes which fails to take account of the visual fields at least roughly in the principal meridians is an incomplete scientific procedure. The instruments I have described enable the busy man to make such examinations easily and quickly.

Whether the cases I have described are various stages of glaucoma simplex, or whether some of them are *sui generis* as suggested by Harry S. Gradle in the paper read before the Academy last year I do not know. Of this, however, I am sure, that I have myself been too slow to recognize a large class of cases that are at least potentially serious.

CONCLUSIONS

1. A cupping of the optic disc and especially a concavity extending to the temporal margin whether deep or shallow is probably pathologic.

2. A large percentage of cases presenting such concavity of the disc will show more or less contraction of the visual fields especially for colors, and many of them one or more other symptoms of glaucoma, especially subnormal sensibility of the cornea, questionable tension or unequal tension of the two eyes, engorgement of the episcleral vessels.

3. An examination of the eyes which fails to take account of such signs is an incomplete examination and must lead to the overlooking of a large number of cases of glaucoma in their earlier and more manageable stages.

DISCUSSION.

DR. LUTHER C. PETER, Philadelphia: The conclusions which Dr. Brown draws from a study of his interesting cases are worthy of special consideration.

The physiologic cup, when pronounced, may border on the pathologic. In fact, I quite agree with Dr. Brown, that a physiologic cup which extends to the edge of the disc, should be regarded as suspicious. When there exists a doubt in the mind of the oculist as to whether the cup is physiologic or pathologic in character, it is always well to regard it as pathologic and to treat it accordingly. It is not necessarily an evidence

that such a patient is, or is likely to become a victim of glaucoma, but some of them at least may have the conditions aggravated by the strain and abuse to which they are subjected in the every-day wear and tear of modern life, or the injudicious use of cycloplegics and mydriatics may add to a tendency which already exists. A small percentage develop a condition which at least simulates glaucoma simplex.

In this connection, the case of a physician, aged 45, who is now under my care, may be cited. He has had vague pains in the eyeballs at times, which he has correctly attributed to approaching presbyopia, which was later relieved by properly prescribed lenses. There is some slight evidence of congestion. The corneas are not anesthetic, pupillary phenomena are normal and the anterior chambers are of average depth. He has a low hyperopic spheric error. The discs show a cupping of about 4 diopters, which includes about four-fifths of the nerve head extending up to the temporal border. Arteries and veins bend sharply over the disc's edge and are lost from view in the receding walls. The right cup is a little deeper than the left. Cribiform plate is clearly seen at the bottom of the cup and is distinctly pale. Form fields are approximately normal in both eyes; blue, red and green fields show considerable shrinking—green out of proportion to the blue and red. In the left eye, the green field is contracted on the temporal side to about 8 degrees. Surrounding the blind spot of the right eye is an indistinct zone for green varying in width from 2 to 4 degrees. This indistinct zone varies with the amount of illumination thrown on the field. Both blind spots show normal outlines for form, blue and red.

The average case of simple noninflammatory glaucoma with cups of this size and depth would manifest field changes far in excess of those obtained in this case. From my experience in a certain number of similar cases, which have come under observation in the past, I am inclined to regard this condition as a disease primary of the optic nerve head which simulates a glaucoma simplex in cupping only—probably a congenital condition increased by the strain of every-day life, and not brought about by the causes usually ascribed to glaucoma.

Without a study of the fields in this particular case, it might be regarded as an exaggerated form of the physiologic cup. The point which I wish to make, however, is this, that although it may be regarded as a physiologic condition, it is a border line case which undoubtedly is beginning to show evidences of a pathologic process, which I feel sure will continue to progress unless the patient is carefully advised as to the abuse and use of his eyes; and even with the greatest amount of care, the probabilities are that the fields will continue to contract and the patient may eventually develop a condition of glaucoma simplex.

One, however, should not overlook the fact that in the stretching process of myopia the fields are apt to show contraction and the nasal fields for many reasons are the first to show such shrinkage. If the cup therefore is deep and extends to the temporal edge of the disc, particularly in the myopic eye, we find a double reason for contraction of the fields.

I am not prepared to agree or disagree with the author's contention that these cases are incipient glaucoma, or that they predispose to glaucoma, because in genuine glaucoma, cupping is the result, not the cause of the glaucomatous process. I can, however, fully agree with him that they should be subjected to the most careful perimetric study, that cycloplegics and mydriatics should not be used, and that the patient should be advised as to the dangers of unnecessary abuse to which the eyes may be subjected.

There can be no dissenting voice to the third conclusion, namely, that an examination which fails to give due consideration to the signs which he has enumerated, is an incomplete examination and leads to the overlooking of glaucoma in its early stages, when something might be done to relieve the condition. In the hurried routine of our office practice, we are apt to neglect examinations by means of the campimeter, which would be of great help in recognizing incipient conditions. It is, however, exceedingly gratifying to note the renewed interest which is being manifested in this direction. Even though an early knowledge may in many instances avail nothing in staying the progress of the disease, it is a source of satisfaction to know that the diagnosis was carefully made and that appropriate treatment was instituted. In not a few instances, on the other hand, early recognition of a beginning pathologic process may yield the happiest results for patient and physician.

DR. E. J. BISSELL, Rochester, N. Y.: I would say that if the group of symptoms which we ordinarily look for are not present and we are to trust to visual field taking in making our possible diagnosis, we must discard the ordinary perimeter and use more accurate instruments. We must test objects of different sizes if we are to detect these cases. This is particularly true in taking the color fields, and we should take the quality of the light into consideration, because this is often the first involvement in a case. The statement has been made by Bjerrum that glaucoma does not exist without affecting the blind spot. The longer I work with improved instruments the more I am coming to accept that view. If the case is myopic you are apt to have an enlarged blind spot. The mentality of the patient is to be considered. Some people are rather slow in giving their response. These are points to be kept in mind.

DR. C. W. HAWLEY, Chicago: On account of the shortness of time for discussion we cannot discuss as we would like, but I wish to call your attention to the fact that the man who has not used the perimeter extensively and taken all the fields for white and color is apt to get into trouble. A few years ago in a number of damage cases in which I was on the stand, I made an extensive study of fields and always found contraction both for color and for white in traumatic neurasthenia. Also we know that the fields get larger and smaller under different irritations. We also must taken into consideration the nervous conditions. So you must be cautious. Also bear in mind that in all cases of glaucoma the extended cupping always commences at the center, so that if we have a physiologic cupping we do not know whether it is extending or not.

DR. S. D. RISLEY, Philadelphia: Of late years I have regretted the uniform use of the term "glaucoma." When a name is mentioned, the thing named rises at once before us as an independent entity. Now so-called glaucoma is not an entity but simply one phase of a syndrome, a part of a pathologic symptom complex, and its use serves to fasten on the mind of the observer a single symptom—increased tension of the globe—to the exclusion of a long chain of pathologic events, both systemic and local, of which the increase of ocular tension is a sequel. For illustration many ocular affections with which we are familiar may be cited; for example, I have many times shown that the readily distensible eye of early life yields to the increase of tension consequent on the intra-ocular congestion and choroiditis due to eye strain or systemic disease, and becomes myopic. I have many times seen cases of hypermetropic refraction in adult life pass over into myopia, in consequence of the protracted increase of tension associated with prolonged uveal disease; as in cases of recurrent iritis or irido-cyclitis, with or without posterior synechia; but much more frequently I have witnessed the syndrome des-

ignated "secondary glaucoma," with serious impairment of central vision and contracted fields. Then there is the extensive group seen in any large clinic of increased tension of the globe as a sequel to certain traumas affecting the anterior segment of the globe showing more or less severe evidence of infection of the globe and also the infectious diseases of the cornea, that is, abscessed, resulting in secondary uveal disease. They often prove refractory to treatment and very many of these eyeballs have been removed and subjected to laboratory study at my clinic and have shed a flood of light on the essential nature of increased tension. With rare exceptions there was found destructive inflammation of the uveal tract; swelling of the ciliary body with synechia both at the pupillary margin of the iris binding it to the anterior capsule of the lens and adhesions of both the ciliary body and root of the iris to the scleral ring. These changes in the uveal tract quite effectually close the excretory channels not only at the angle of the anterior chamber, but at the vena vorticiosa and in the region of the optic nerve. The choroid is found tightly attached to the sclera and the optic nerve cupped as a sequel to the inevitable rise of intraocular tension, due to closing of the excretory ducts. Briefly the uveal condition of which the rise in tension is a sequel may be occasioned by purely local condition or a local expression of systemic disease of the toxic or infectious types and no treatment is efficient for its relief which is not based on these considerations.

DR. BROWN (closing discussion): Following the reading of Dr. Wells' paper on the use of the Haitz charts and his suggestion of using stereoscopic vision and binocular fixation, I have used a concave copper disc with a perforation of one-eighth inch held before the eye under examination, which I find of great use in helping the patient keep his eyes where they belong.

I am grateful to the gentlemen who have discussed my paper and from whom I have learned much. I was glad to hear Dr. Risley say it is increased tension that makes trouble with young people with beginning myopia, and I believe that tension is a relative matter. What may be pathologic tension in one patient is not so in another, so it seems to me the tonometer has a very limited value.

The essential thing is that eye strain is the cause of both myopia and chronic simple glaucoma.

THE ADAPTABILITY OF THE PHORO- OPTOMETER STEREOSCOPE FOR THE HAITZ AND BISSELL CHARTS

DAVID W. WELLS, M.D.; F.A.C.S.

BOSTON

In using Haitz charts, I at first followed Haitz instructions, using an ordinary Holmes stereoscope marking on the same the distance at which the chart should be viewed. It was soon found that holding the stereoscope was tiresome for the patient, who kept it quite unsteady. As I make constant use of the phoro-optometer I found it was much more satisfactory to make the stereoscope to order for each patient.

I use for each eye $+5.25$ with the chart at 19 cm. In the ordinary use of the phoro-optometer stereoscope $+10$ is employed with the cards at 10 cm. This makes each prism diopter = deviation of 1 mm. With $+5.00$ at 20 cm. and practically the same at 19, each prism diopter = deviation of 2 mm. With the B3 card the natural fusion distance is determined and sufficient prism introduced to make lines cross at 8, since Haitz charts have a separation of 8 cm. For example: should the red line cross at 5 this means that 5 cm. is the natural fusion distance. To secure the crossing at 8, fifteen diopters more is required since 30 mm. more deviation is needed and at 20 cm. each prism diopter = 2 mm.; $30 \div 2 = 15$. If the patient happens to converge, more prism will be needed, the exact amount being determined by turning the rotary prisms. The patient should wear his refractive correction, and if he be presbyopic, his reading glasses, or the necessary amount added to the $+5.00$.

The stereoscope is now ideal for mapping a central scotoma and at the same time it has been demonstrated that the patient has binocular vision sufficient for the test. Since visual acuity is often poor and fusion faculty of low grade, it was found that the lines of the B3 card were too fine to be easily seen by many patients. I have therefore drawn upon the back of the chart a modification of B3 with heavier lines.

In using the Bissell Blind Spot Chart it is necessary to get a wider field. This is easily obtained by commencing with the

+5.25 decentered out 10 mm. wider than the patient's P.D. and substituting for the revolving prisms, after the necessary amount has been determined, plain prisms from trial case. This increased distance of the lateral part of the chart changes the perfect correspondence of squares to degrees of the arc; the error being $\frac{1}{4}$ degree in the whole size of a normal blind spot; but, if one wishes to be more exact the chart can be bent by having a third support, so that the ends and the center will be the same distance away. (See Appendix.)

Lloyd's Slate (described in the *Ophthalmic Record*, August 1917) is a combination of the Haitz and Bissell charts and therefore suffices for both purposes. Dr. Lloyd kindly sent me one which I have found very satisfactory. The color of the cross lines is much more subdued than the other charts of American manufacture which I have seen, and is therefore less confusing. If one is not fortunate enough to possess the original Haitz, he will find this a decided improvement even for central scotomata, while for the blind spot measurement it shows at a glance the relation to the macula, and the normal area according to Gradle.

The writer suggested to Lloyd and Bausch & Lomb, the making of record slips duplicating the slate. These suffice for recording either central or paracentral scotomata or blind spot measurements, for one or both eyes, and always maintain the relative positions. The data suggested by Bissell are printed on the back. One may mark on the slate with chalk, as Lloyd suggests, and transfer the record later, but I have found it quite satisfactory to have my assistant stand behind the patient—slip in hand—watching the point reached by the object when the patient first sees or first loses it, and mark it immediately on the slip. In this way the permanent record is finished as soon as the test is completed.

Those who do not use the phoro-optometer will probably prefer to get the special wide angle stereoscope (Bausch & Lomb). But the method described does not necessitate the patient's moving from his chair, dispenses with a new instrument and gives one a knowledge of the patient's fusion faculty, which should be investigated before attempting the test. The only expense involved is the central aluminum screen, the cost of which is trifling. Moreover, the writer still lives in hopes that more of his colleagues will recognize the value of the phoro-optometer stereoscope in fusion training and the cultivation of adduction. This adaptability for the Haitz and Bissell charts is an additional reason for the adoption of this wonderful instrument.

In December 1916, there was published in the *Journal of Ophthalmology, Otology and Laryngology* a translation of Haitz' description of his charts. Of this there are a few reprints left which will be sent to those desiring them.

APPENDIX—DISTINCTION BETWEEN DEGREE AND PRISM DIOPTR.

The author must protest against the very common mistake of confusing the terms Degree and Prism Diopter. A *degree* ($^{\circ}$) is $1/360$ of the arc of a circle, and should be restricted to Perimetry, the measurement of Heterotropia, and Stereoscopic Kempimetry.

The unit of the *prism diopter* is a prism which deflects a ray of light 6 cm. at 6 m. Heterophoria is therefore measured in prism diopters. According to Prentice's rule, (*Ophthalmic Lenses & Prisms*, 1917, p. 49), the prism diopter = tangent of $34'22''$, figured as follows:

$$\begin{array}{rcl}
 \text{Tangent} & \dots\dots\dots & 1\Delta = .01 \\
 \text{Nearest smaller in table is—} & & \\
 \text{Tangent} & \dots\dots\dots & 0^{\circ} 30' = .008727 \\
 \hline
 \text{Difference} & \dots\dots\dots & = .001273 \\
 \text{Nearest larger in table is—} & & \\
 \text{Tangent} & \dots\dots\dots & 0^{\circ} 40' = .011636 \\
 \text{Nearest smaller in table is—} & & \\
 \text{Tangent} & \dots\dots\dots & 0^{\circ} 30' = .008727 \\
 \hline
 \text{Difference} & \text{for } 10' & = .002909
 \end{array}$$

Dividing the whole difference by difference for $1' =$ minutes to be added to $30'$,

$$\begin{array}{r}
 .001273 = 4.37' + 4' 22'' \\
 \hline
 .0002909
 \end{array}$$

$$\therefore \text{Tangent } 1\Delta = 0^{\circ} 30' + 4' 22'' = 34' 22''.$$

A 1° (degree) apex angle prism of glass, index 1.53, deflects a ray of light $31' 48''$ (*Prentice: Ophthalmic Lenses*, 1900, p. 108), so that for ophthalmologic purposes the prism diopter has become the prism unit and deflects a ray of light about $\frac{1}{2}^{\circ}$ (degree).

THE STEREOSCOPIC TANGENT OF 1°

Haitz has calculated his chart for $+5.25$ as this is the strength used in the standard stereoscope. At the focal distance 19 cm., this makes each square 3.3 mm. If o. u. $+5.00$ is used at 19.8 cm. he says each square is .16 mm. too small. This is easily figured by the principal of similar triangles as shown by Peter (*Principles and Practice of Perimetry*).

From tables we obtain tangent of $1^\circ = .017455$,

1 : .017455 = focal distance : tangent required.

.017455 x 18.2 = .317681 cm. = 3.17681 mm. = tangent at 18.2 cm.

.017455 x 19 = .331645 cm. = 3.31645 mm. = tangent at 19 cm.

.017455 x 19.8 = .345609 cm. = 3.45609 mm. = tangent at 19.8 cm.

.017455 x 20 = .349100 cm. = 3.491 mm. = tangent at 20 cm.

.017455 x 21 = .366555 cm. = 3.66555 mm. = tangent at 21 cm.

This corresponds exactly with the result obtained above for 19 cm., but the difference between the tangent at 19 cm. and 19.8 cm. is .139 mm., and not .16. Since Haitz says the .16 mm. is "Plainly negligible," this error is of no account. In order to be more exact the author had made a very perfect pair of $+5.25$ toric lenses. This takes care of the central measurements at 19 cm., but the most lateral portion of the blind spot is almost 1 cm. farther away, that is 20 cm., at which distance the square is .17 mm. too small. If the blind spot has a lateral diameter of 5° (degree) ($4^\circ 54'$ Gradle), the whole error would be $5 \times .17 = .85$ mm. As the square is 3.3 mm. $.85 = .25^\circ$ (degree). This is, the blind

3.3

spot would be mapped $\frac{1}{4}^\circ$ (degree) larger than reality. This is of course too slight to be of any moment, but if one wishes to be more exact the chart can be bent by having a third support so that the ends will be just the same distance as the center.

POINT FROM WHICH TO MEASURE RADIUS

There is one important item about which the author is unable to agree with the authorities and that is the *point from which* this 19 cm. should be measured. Haitz says: "Inasmuch as the focal distance is reckoned from the side of the lens turned toward the slide, the latter is to be brought to such a position that the middle of both pictures stands at about 18.8 cm. *from the bottom of the side of the lens turned toward the nose.*"

Dr. Bissell¹ says: "The card carrier should be just within the focal plane of the lenses, at 18.8 cm. In this position the rulings of the card have a normal 1° angle if the center of rotation of the eyes is at 25 mm. from the *anterior* surface of the lenses," but he furnishes no means of obtaining these data. Presumably this means that the lenses should be 13 mm. in front of the corneæ, and omitting the question of rota-

¹Exhibition of a special wide range stereoscope for the Haitz & Bissell Tests. Acad. Ophthal. & Oto-Laryn., Pittsburgh, October, 1917.

tion, the 18.8 cm. is measured from the *anterior* surface of stereoscopic lenses.

Dr. Bissell refers to Mr. Max Poser, of Bausch & Lomb, as his authority, but notwithstanding his eminence as a physicist, it is impossible to regard this statement as satisfactory.

In constructing a wide angle toric lens it may be necessary to assume it will stand 25 mm. in front of the center of rotation in order that the aberration may be corrected as the eye turns, but he has adopted Haitz' point from which to measure the radius, namely, the anterior surface of the stereoscopic lenses.

Moreover he furnishes no means of determining the center of rotation, and with a hooded stereoscope it is not possible to observe or measure the distance of the lenses from the corneæ, which is the only way of *approximating* the center of rotation.

In constructing a tangent screen Dr. Peter (*ibid.*), says that radius is to be measured from the front of the cornea.

THE NODAL POINT

To be exact it seems to the writer that the *nodal point*, being the point through which pass all rays which enter into the formation of the image, must be the apex of both similar triangles, *and must, therefore, be the point from which to measure radius.*

In the schematic eye this point is usually given as 7.3 mm. for anterior and 7.6 mm. for posterior behind the cornea. The addition of a +5.25 will carry this point forward, the exact amount depending on the form of the lens and its distance from the eye. If the lens be toric with a base curve of -6.00 and an anterior curve of +11.25, and if it be situated at the standard distance 13 mm. in front of the cornea, the nodal point of the whole system, lens and schematic eye combined will be from 2 to 3 mm. farther forward, approximately 5 mm. behind the cornea.

Thus it appears that the nodal point is about 5 mm. + 13 mm. = 18mm. back of the stereoscopic lenses. This is practically 2 cm., so that the Haitz chart with the 3.3 mm. square should be placed 19 cm. — 2 cm. = 17 cm. in front of the stereoscopic lenses to secure the nearest approximation to a one degree equivalent.

Reference to the table on page 112 shows this error would make each square $3.66 - 3.31 = .35$ mm. too small. This error would make the normal blind spot $\frac{1}{2}^\circ$ too large but is easily corrected by adopting 17 cm. as the distance of the chart from the back side of the stereoscopic lenses. This is *equivalent to measuring the radius from the nodal point.*

But this calculation is for a schematic eye. "The nodal points differ in various eyes according to their refractive power and such differences may even exist in two eyes, having the same degree of ametropia, so that punctilious precision as applied to their positions does not seem possible of attainment.

"Besides with the use of a stereoscope there is always an error of parallax incurred due to the nodal point and the point of prism refraction not being made to coincide. This parallax is least when the eye is closest to the apex edge of the prisms; and even were the cornea in actual contact with the prism surface, the nodal point would still not be at the point of refraction of the prism. Hence the discrepancy between the plottings of the scale and the fundus will ever be present through the use of a stereoscope." (Chas. F. Prentice; personal communication.)

It is possible that the improved wide angle stereoscope may eliminate this error of parallax, but until the radius is measured from the nodal point, and the stereoscopic lenses are at a fixed distance, say 13 mm, in front of the corneæ, it must be less exact than the phoro-optometer stereoscope.

Perimetry with or without a stereoscope does not correspond exactly with degrees of the retina. However, this limitation does not materially detract from the practical value of stereoscopic perimetry, as the error need never exceed $\frac{1}{4}^{\circ}$ (degree) if the 19 cm. be measured from the schematic nodal point of the combined system, and one would hardly presume to diagnose abnormality unless the enlargement of the blind spot were as much as 1° (degree). Moreover, the comparison of successive examinations made under similar conditions will show any change that may have occurred, that is, the error will be constant.

At the present writing the phoro-optometer stereoscope furnishes the most exact means of reproducing similar conditions. The lenses may be brought as close to the lashes as possible without touching, the pupillary distance of the lenses and the exact amount of extra prism exhibited can be recorded.

A SPECIAL WIDE ANGLE STEREOSCOPE FOR USE
WITH HAITZ AND BISSELL CENTRAL
SCOTOMA AND BLIND SPOT
TESTS

ELMER JEFFERSON BISSELL, M.D.
Fellow of the American College of Surgeons
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It is coming to be generally recognized that if visual field work is worth doing at all it is worth doing as accurately as possible, and to attain accuracy it is necessary to select the instrument and method best adapted to the special data desired. With every degree that we approach the fixation point the element of accuracy becomes more important and the difficulty of securing it increases. Dr. Peter, in a paper before this society last year, made a statement which needs to be emphasized—"the boundaries of the projected field are necessary, but an analysis of the character of the preserved field is equally important." I would add it is usually more important.

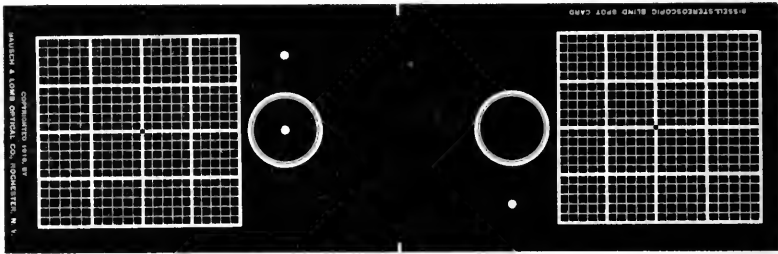
With all instruments and methods it is desirable to secure as easy and definite fixation as possible. With the Bjerrum screen the author's two-point alignment instrument is of great assistance in maintaining fixation. In a large group of very important cases the stereoscope makes available the great advantage of single binocular fixation, the value of which has not yet been fully appreciated. Haitz gave to the profession seven stereoscopic cards which I believe have no equal for accuracy in detecting small central and paracentral scotomata within 10 degrees of the center of fixation. The author presents a new blind spot card which supplements the Haitz set and thereby increases the range of usefulness of the stereoscope in visual field testing.

Even when there is a central scotoma in each eye not larger than 3° the stereoscope may still have advantage over any monocular method. This was pointed out by Haitz when he showed that the border of the stereoscopic figure may be fused and the tests carried on satisfactorily.

I have had the Bausch and Lomb Optical Company make a wide angle stereoscope which has the elements of a scientific instrument for this work.

ADVANTAGES OF INSTRUMENT

1. Clear definition over the entire field, including the area of the blind spot, is secured with $+5.25$ D. lenses, 36 mm. in diameter, of the meniscus type, similar to punktals, corrected for astigmia of oblique pencils over an area of 30 degrees from the center. The focal distance of these lenses is 19 cm. In front of each lens is a diaphragm with a horizontal oblong opening.



Bissell stereoscopic blind-spot card.
(1/7 full size.)

This restricts the field of vision to the test card and cuts off any motion of the hand in moving the test object. If the distance ametropia is corrected, the accommodation is inactive during the test. A receptacle is provided into which the correcting lenses or any prisms may be inserted.

2. The instrument is adapted to eyes of different pupillary distance. This is secured by the special type of lenses of large size with a mechanical interpupillary distance of 80 mm. With this arrangement it is not necessary to have the lenses adjustable laterally except to correct small amounts of exophoria or esophoria, as fusion is secured through any portion of the lenses. The lenses are decentered to secure 2.6 prism diopters and can be rotated to correct any moderate amount of hyperphoria. With these mechanical conditions the optic axes are practically parallel during the test.

3. The card-carrier is adapted to receive all sizes of cards. The blind spot card which is 8.5 cm. longer than the ordinary stereoscopic card can be used with this instrument. The card-carrier can be moved along a graduated rod, but ordinarily should be just within the focal plane of the lenses, at 19 cm. In this position fusion is perfect and the rulings on the card have a 1 degree angle.

4. The stereoscope can be tilted at any desirable angle and is

mounted on a firm base. This prevents the unsteadiness associated with a hand stereoscope.

5. Central color testing is very satisfactory with it even by artificial illumination if a special desk lamp with a "linolight" bulb and a "daylight" filter is used.

The Haitz cards are now so familiar to oculists that they need not be described. The author's blind spot card is 8.5 cm. longer than the Haitz and over a space of about 6 square cm. at each end of the card, where the area of the blind spot would be, there are faint grayish squares of about 3.3 mm. printed on a black cloth, slatelike surface. These squares vary in size on the tangent surface so as to represent 1 degree visual angle if viewed through a properly constructed wide angle stereoscope, such as just described. The stereoscopic portion of the card consists of two grayish rings 8 cm. apart and about 3 cm. in diameter. In the center of one ring is a whitish 2 mm. spot. There is also a 2 mm. whitish spot above one ring and a similar spot below the other so that when viewed in the stereoscope there can be no question that fusion is secured if the three dots are seen in a perpendicular plane and only one of the rings appears. Fixation is more definitely maintained with these simple rings and dots than by a more complicated stereoscopic figure.

DIRECTIONS FOR TESTING

The patient's distance ametropia must be corrected if of an amount sufficient to reduce the vision. He should sit near a window (preferably a north light) and be told to steadily look at the white spot in the center of the circle. The test objects to be used are white, blue, red and green round vulcanite discs 1.5 and 3 mm. in diameter mounted on a thin metal rod. If the vision is good, the 1.5 mm. white disc is usually preferable, but in testing with colors the 3 mm. is better. These are slowly moved over the area marked in 3.3 mm. squares and first the four cardinal points of the blind spot determined and then the 45 degree points. The normal blind spot will be from 13 to 15 degrees from the fixation point and will occupy about $5\frac{1}{2}$ of the squares horizontally and $7\frac{1}{2}$ vertically (each square represents 1 degree). The test object should be moved both from visible to invisible and invisible to visible. Having the small squares as guides the points as discovered can be marked with chalk upon the slatelike surface and later transferred to a Bissell record card which is ruled the same as the test card, or the examiner can at once indicate the points on the record card as

he proceeds with the testing. I have had printed on the back of the record card the various factors which may influence the test such as size of pupil, method of signal, character of light, size of test object, refraction, etc. A check mark, measurement or explanation can be quickly entered at its appropriate place and this information is invaluable, not only for future reference and comparison with other tests, but in interpreting the test to which it relates. The same test objects and record cards can be used in the Haitz tests.

ARTIFICIAL DAYLIGHT ILLUMINATION FOR PERIMETRIC STUDY AND GENERAL OFFICE USE

LUTHER C. PETER, A.M., M.D.

PHILADELPHIA

The need of artificial daylight illumination has long been felt, not only for the purpose of lighting up test cards and to obtain uniform results in perimetric work and color studies, but in many instances in order to obtain satisfactory and comfortable lighting for the average professional office in large cities.

When a good northern exposure with adequate window facilities can be obtained, the problem of office illumination is easily and satisfactorily solved. This, however, is the exception, rather than the rule, and even with these conditions fulfilled, there still remains the question of the test card illumination and the proposition as to how to obtain uniform results in perimetric work under variable weather conditions.

Much study has been given to the reproduction of daylight during the past ten years and Dr. Herbert E. Ives, of the Photometrical Laboratory of the U. G. I. Company, Philadelphia, and Dr. Henry Phelps Gage of the Corning Glass Works, Corning, N. Y., have contributed largely to the perfecting of methods to accomplish this end. It is needless for our purposes to enter into a discussion of the intricacies of the problems encountered in evolving the practical and satisfactory devices which are now offered to us. It is pertinent, however, to analyze our peculiar needs and the substitutes for daylight which are most suitable for our purposes.

For office illumination effective artificial light is not always either satisfactory or comfortable. When simply the question of illumination enters into the problem one of the ordinary forms of electric lights or the Welsbach burner, when properly safeguarded so as to remove direct glare, may suffice. This form of illumination, however, will rarely furnish the comfort to the eyes equal to that which is provided by the yellow of the sun's rays, blended with the blue of the sky and filtered through a rather homogeneous atmosphere. Most of these artificial lights, when subjected to spectrum analysis, will show an excess of red, green and yellow. The problem, therefore, which confronts the

chemist is the blending of a glass filter so as to reduce or absorb the excess colors in order to produce a spectrum analysis which is similar to that of daylight. This is a scientific problem which we as physicians must relinquish to the physicist and to photometric laboratories.

What are our particular needs? For perimetric and color studies, uniformity and constancy are important factors. Daylight will vary at different times of the day and from day to day under changing atmospheric conditions. If, therefore, we can obtain artificial illumination which approximates the spectrum analysis of the sunlight, there can be no objection to its use. On the contrary, if such a light can furnish uniformity and constancy in saturation of colors it must commend itself to us.

In the industrial world artificial illumination is employed daily in the careful analysis and color matching of dye houses and textile mills, and if satisfactory in so crucial a test, it should be suitable for our professional purposes. The distinct advantage to be gained is to be able to examine patients at any time under uniform conditions and under conditions which can be obtained in any office.

In the selection of a suitable light, one factor at least must be considered, namely, the cost of installation and maintenance. According to Gage (*Journal of Franklin Institute*, May 1914), an incandescent black body at 5000 C should furnish a spectrum distribution of the intensity of daylight, but "such a temperature," he says, "is beyond present means." The passing of electric currents through a rarefied gas (a carbon dioxide vacuum tube, sold in the market under the trade name of the Moore tube) has been extensively practiced commercially and with considerable success. The cost of installation and maintenance, however, is prohibitive. An absorbing screen from the standpoint of economy and efficiency has therefore proved to be the most practical method of obtaining artificial daylight.

Some months ago, through the courtesy of a local firm in Philadelphia, the Corning "daylite" screen was placed on my desk for experimental study. The light emitted was so free from glare and so much in its general effect like natural daylight that I was prompted to investigate its qualities. In common with all forms of screens devised for a similar purpose, the light furnishes only 15 per cent. of efficiency, the balance being absorbed by the filter so constructed as to reduce the blue, red and yellow to normal daylight values. This, however, is the only objection.

The Corning Glass Works, who manufacture it according

to the method evolved by Dr. Henry P. Gage, claim that in its spectrum analysis, it approximates normal daylight. This claim has been verified by other physicists.

For my perimetric studies, I have had in use a 75 watt Mazda lamp covered by the "daylite" screen protected by a deep reflector so as to prevent glare. It furnishes an evenly distributed illumination to the campimetric surface which gives one the sensation of good daylight exposure and is the most satisfactory form of artificial illumination that I have thus far employed. The illumination of the test card is softer and whiter than by Mazda lamps covered with frosted glass. For desk use the light is ideal.

I have no desire to appear in the rôle of an advertising agent for the output of any commercial house, but as we are dependent on the product of some photometric laboratory, I do not hesitate to recommend to the profession this "daylite" lamp or screen as being admirably adapted to our special needs. Furthermore, with uniformity of illumination obtained at a nominal cost, there can be no objection to concerted effort on the part of the profession to do their perimetric work under the best possible conditions with at least an attempt at standardization of methods. From a scientific point of view, there can be no objection to an artificial illumination which will stand the test of spectrum analysis.

We have much to learn in qualitative and quantitative perimetry. Quantitative studies have been fairly well worked out by the means at hand, and our knowledge has a fair degree of accuracy even without an attempt at standardization of methods and of illumination.

Qualitative studies which, after all, are the studies of real value, have not been developed to the same extent. Standard illumination has been lacking. Color thresholds have been studied under all degrees of illumination, notwithstanding the fact that there is a vast difference between studies of the light adapted eye and the scotopic or dark adapted eye. Examinations made in a subdued light, or in so-called twilight, will not yield the results of the maximum photopic vision. Standard illumination, therefore, which can be controlled, will not only aid materially in obtaining uniform results, but will help to develop valuable scientific information in qualitative perimetry.

A SATISFACTORY OPERATION FOR MUSCLE SHORTENING OR ADVANCEMENT

MAJOR WALTER B. LANCASTER, M.R.C., U. S. ARMY.

BOSTON

The final solution of the problem of muscle shortening or advancement has not been reached—is not likely to be reached. As Poincaré has said, "We do not have two classes of problems in science, those that are solved and those that are not, we have only problems *more or less* solved." I say this that you may not think I am attempting to utter the last word on advancement operations. My object has been to find among the multitude of proposed methods the most satisfactory procedures.

For anesthesia I prefer local to general. Some cases will be found who are too excitable and uncontrollable for satisfactory operation by local anesthesia. The number of these will be greatly reduced if the surgeon really does not hurt the patient and if adequate sedatives are administered some hours or days beforehand. Superficial anesthesia is, of course, easily obtained by dropping 2 to 4 per cent. cocain into the conjunctival sac. This makes the conjunctival incision and isolation of the muscle painless; but when the muscle has been raised on the hook, very little pulling will suffice to cause pain. Once the patient has been hurt, he is made nervous and apprehensive and becomes harder to manage. To operate satisfactorily, the surgeon must be able to make any manipulations he may find necessary without hurting. If the solution of cocain or other anesthetic is injected subconjunctivally, the edematous infiltration, even if the eye is massaged, is sufficient to interfere with the operation. The most satisfactory way is to inject the novocain or cocain solution beneath the capsule of Tenon, pushing the needle in about 20 mm. alongside the muscle. One, or at the most 2, c.c. of a 1 per cent. solution of novocain, or a smaller quantity of one-half per cent. cocain, always with a small amount of suprenin or adrenalin added, will produce so complete anesthesia that when the muscle is lifted on the hook and pulled on no pain will be felt if manipulations are reasonably gentle. When the patient finds that no pain is felt, he quiets down and remains a satisfactory subject; but once a real twinge is felt, the patient becomes aroused, nervous, apprehensive and hard to manage.

The most satisfactory conjunctival incision in my experience is a longitudinal straight incision from near the cornea to near the canthus (Fig. 1). This gives a satisfactory exposure of the

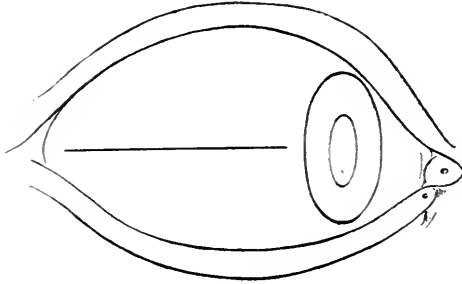


Fig. 1.

field, provided the two lips of the wound are retracted either by the assistant or by a temporary suture through each with a forceps snapped on each as a weight. The line of incision is parallel to the chief blood vessels, and so the hemorrhage is less, and the nutrition less interfered with during healing. These are very unimportant considerations, because the hemorrhage and healing usually give no trouble. The chief advantage is in the cosmetic effect. With the commoner more or less transverse incisions (Fig. 2), there is too often an ugly blemish, lasting

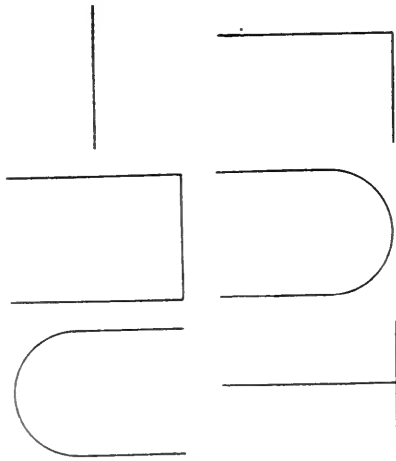


Fig. 2.

for weeks or even months, where the conjunctiva covering the site of operation is thickened, hyperemic and somewhat puckered, in marked contrast to the smooth adjoining conjunctiva (Fig. 3).

Sometimes this blemish is visible for years and may never disappear. It is, therefore, an important consideration. This blemish is not an inevitable consequence of the various transverse

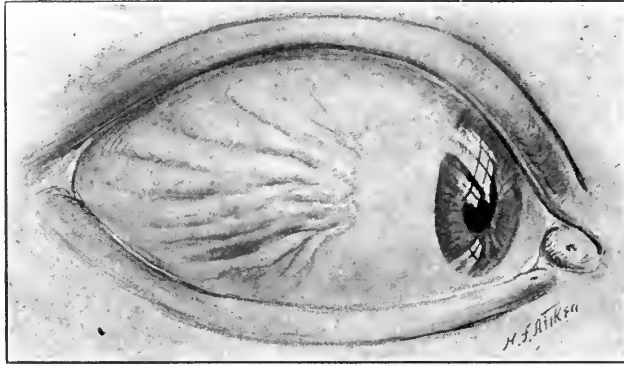


Fig. 3.

incisions or flaps. It is, however, much more difficult to secure a satisfactory, smooth and good looking scar with that form of incision. Sometimes the operator has been known to cut too deeply in making his conjunctival incision because he picked

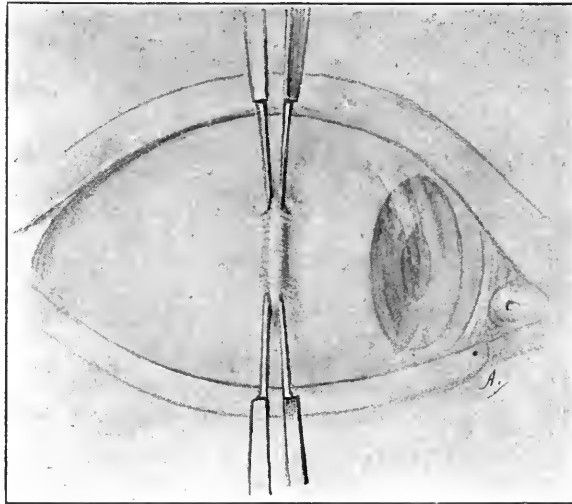


Fig. 4.

up too much tissue with his forceps including muscle as well as conjunctiva. If the incision is longitudinal, no harm is done, because it is parallel to the muscle fibers; whereas, such an inadvertent cut across the muscle fibers would be a serious

mistake. Hence, if a transverse incision is chosen, it is safer to make it anterior to the tendon insertion.

To make this longitudinal conjunctival incision, the surgeon seizes the conjunctiva about as far from the cornea as the insertion of the muscle and near its upper or lower border, with one blade of the forceps nearer the cornea and one nearer the canthus, thus raising a transverse fold. The assistant seizes this fold in the same way near the opposite border of the muscle 4 mm. from the surgeon's forceps (Fig. 4). This fold is cut with the scissors and the incision extended toward the cornea and toward the canthus. Without releasing the forceps, each flap is undermined as far as the full width of the muscle (about 10 mm.), or approximately the width of the cornea (Fig. 5).

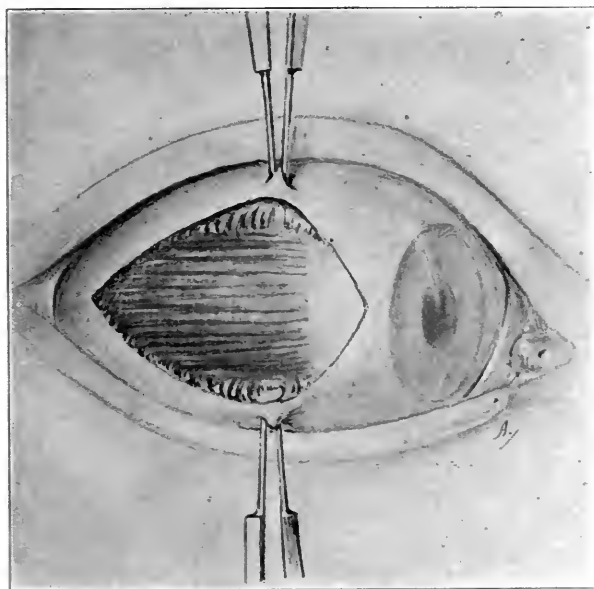


Fig. 5.

The surgeon releases his hold of the conjunctiva and picks up a transverse fold of capsule of Tenon just beyond the margin of the muscle, where it is exposed by the assistant, who still retains his hold of the conjunctiva (Fig. 6). The capsule is cut, making a longitudinal opening. Still holding the fold of capsule in his forceps, the surgeon lays aside the scissors and takes a medium-sized muscle hook, which he inserts through the opening in the capsule. If the opening has been properly made, the hook is inserted with great ease and passed under the muscle until

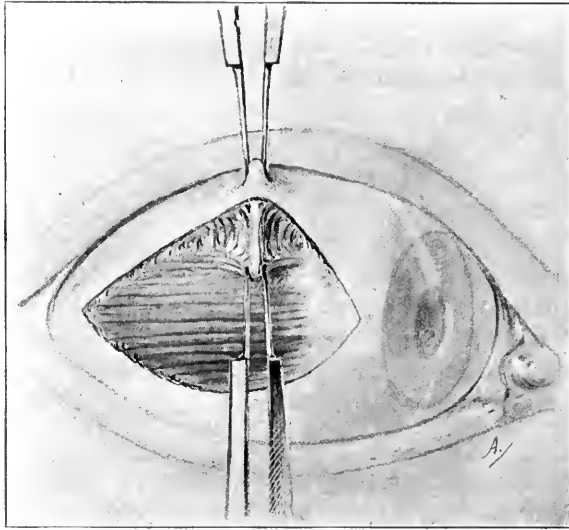


Fig. 6.

its point is made to lift the capsule at the opposite border of the muscle (Fig. 7). The assistant lifts the conjunctiva away

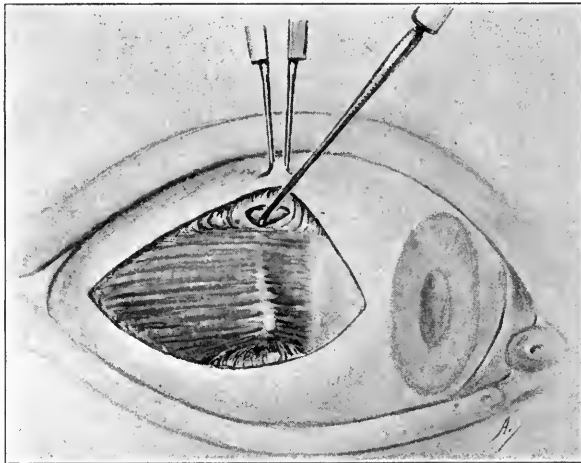


Fig. 7.

from this border of the muscle, and the surgeon transfers the hook to the left hand and with the scissors snips an opening in the capsule over or alongside the hook, permitting it to come through (Fig. 8). Before enlarging these two openings, thus isolating the muscle, make sure that the entire width of the

muscle is included. Not infrequently, the incisions, one or both, are made too near the median line of the muscle and so do not include all the fibers. Remember that the width of the muscles

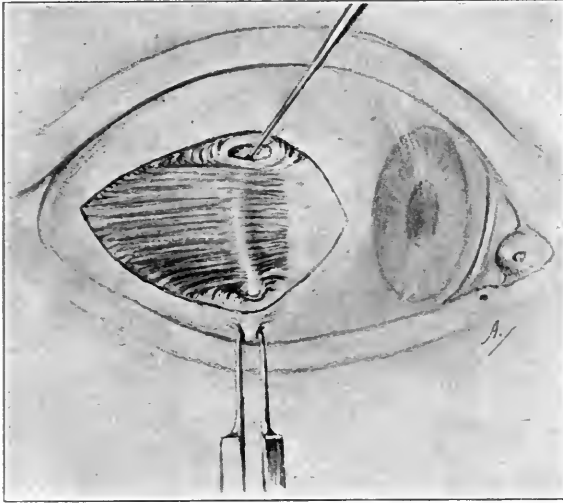


Fig. 8.

is nearly equal to the diameter of the cornea, and not one-half that width, as often shown in illustrations.

The two incisions through the capsule should be prolonged by cutting with the scissors, rather than by introducing two hooks and tearing (Figs. 10 and 11). How long to make these incisions depends on how much the muscle is to be shortened.

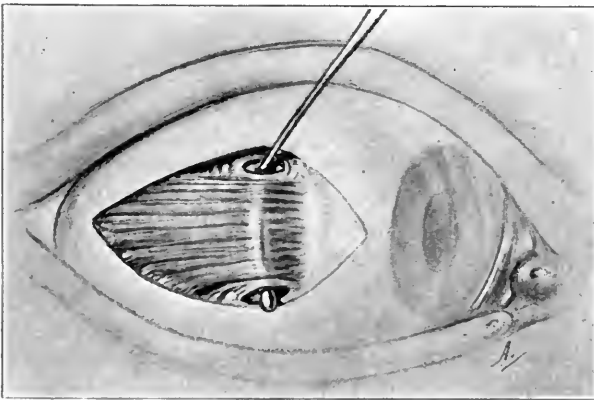


Fig. 9.

One can stop here and tenotomize the antagonist in the usual way, or this can be done later.

The suture through the muscle to be shortened is next placed. The most satisfactory hold is obtained by a whip stitch. A double armed silk suture, not too fine, is used. One needle is passed through the muscle from the outside toward the scleral surface about 2 or 3 mm. from one margin of the muscle and as far back from the insertion as is thought necessary to obtain the desired shortening (Fig. 12). The same needle is passed

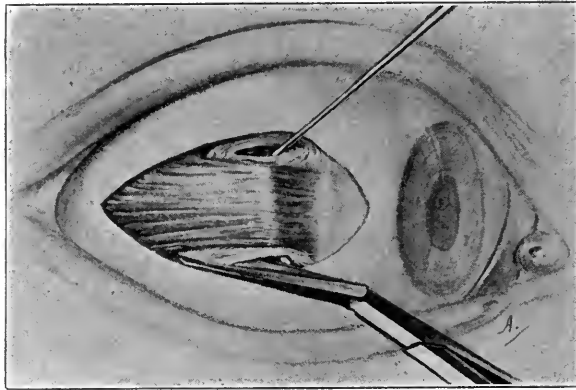


Fig. 10.

again just back of the point where it was inserted the first time, thus surrounding a bundle of fibers about one-quarter to one-third the width of the muscle with a whip stitch. In the same way, a whip stitch is passed around the corresponding bundle of fibers along the other border of the muscle (Fig. 12). The operator now has a secure hold on the muscle, and can cut the tendon.

An alternative method is to seize muscle with Prince's forceps near insertion and cut it off before inserting the suture. This can then be very easily done because the muscle can be held up away from the eyeball where it is easy to get at. If the patient is told to look toward the side of the antagonist the muscle will be relaxed and more easily drawn up by the forceps. It is well to leave a stump of about 1 mm. rather than cut the tendon clean off close to the sclera. This stump affords a secure hold for the forceps in fixing the globe when passing the stitches in the sclera later on. It also affords an easy anchorage for an additional security stitch to hold the muscle. This should be placed next.

Use a double armed suture of silk, not too fine. It may be of a different color from the first one. Pass the needles from behind and beneath the stump forward through the insertion, one emerging 3 mm. from the upper margin, the other 3 mm. from the lower margin of the tendon and a little nearer the cornea than the line of insertion. The loop of the suture is on the scleral surface of the tendon (Fig. 13). Next, pass

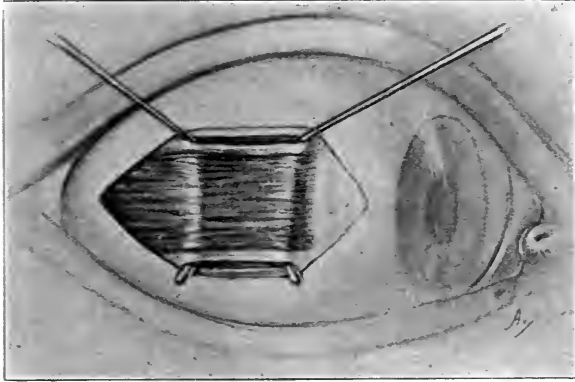


Fig. 11.

the needles through the muscle from the scleral surface outward about 3 mm. farther back than the whip stitch and about 3 mm. from the margin of the muscle (Fig. 14). Then pass the needles

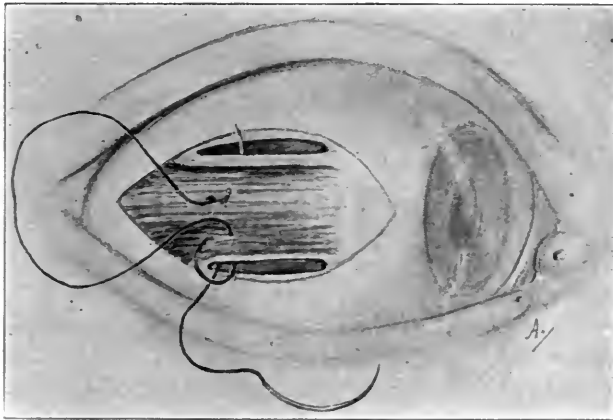


Fig. 12.

through the conjunctiva about 2 mm. from the margin of the incision and about as far from the cornea as the insertion of the tendon. When the muscle has been pulled forward and

tied in place by the first or whip stitch, this security stitch will be tied, thus binding the muscle down on the tendon stump and closing the conjunctival incision at the same time. Meantime, lay these threads one above and one below the eye on the sterile cloth with which the face and head are covered. Next, pass the needles of the first suture, which is attached to the muscle, through the superficial layers of the sclera near the cornea. The most satisfactory way to do this is to fix the globe with a fixation forceps, which securely grasps the tendon stump left for that purpose. Enter the needle between the tendon insertion and the cornea, preferably near the cornea, where the sclera is thicker and where an accidental penetration of the eyeball would be least disastrous. Push it toward the cornea, that is, away from the tendon, nearly parallel to the direction of the muscle fibers (Fig. 15). When the needle is pushed in this direction

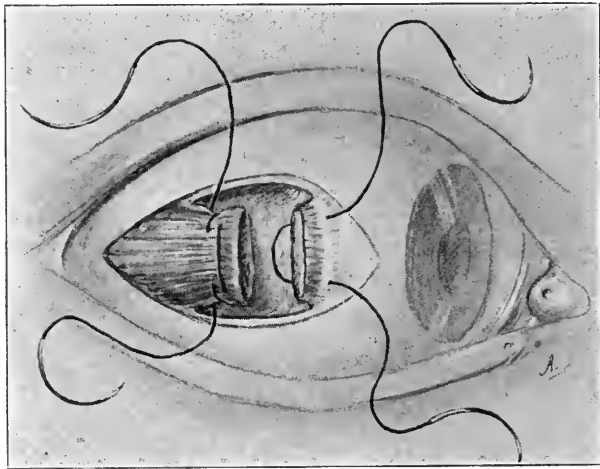


Fig. 13.

and the eyeball held firmly by the tendon stump, there is no tendency for the eyeball to roll around, and it is much easier to make sure that the needle goes through a few superficial fibers of the sclera, getting a firm anchorage without going too deep and without tearing out from not going deep enough or from twisting, rolling movements of the eyeball. The most satisfactory place for the needles to emerge is through the conjunctiva about 1 mm. from the corneal margin, and one 2 mm. above and the other 2 mm. below the original longitudinal incision in the conjunctiva. When the suture is tied, the knot will be outside on the surface of the conjunctiva.

The most satisfactory way to tie this knot is to have the assistant seize the globe by the stump of the tenotomized antagonist with one hand and the end of the tendon to be advanced with the other, and while rotating the globe toward the muscle to be advanced with the first hand pull the muscle forward with the other. The surgeon pulls gently on the ends of the suture, taking up the slack, and then more firmly until the muscle is brought as far forward as is desired, when a single knot is tied. Unlike the Reese, Verhoeff and other similar methods, it is not necessary, in order to get a surely holding suture, to pull it forward until the point where the suture is attached to the muscle is right up to the point where the suture is attached to the sclera, since the security stitch will tie the muscle down on to the sclera.

The eyes are now inspected, and if the effect is satisfactory the second half of the knot is tied firmly. If not enough effect has been produced, the assistant again grasps the muscle and the stump of the antagonist, and rotates the eye toward the muscle while pulling the muscle gently forward, while the surgeon pulls the suture more tightly. If the effect is insufficient even when the point where the stitch is fastened to the muscle is drawn close up to the point where the stitch enters the sclera, there is no use in pulling any more. The suture was not applied to the muscle far enough back, and must be done over again, a matter of a very few minutes and very little trouble when the anesthesia is satisfactory.

After the whip stitch is tied, the security stitch is tied. Usually, this suffices to close the conjunctiva. Good apposition of the edges favors prompt union and good cosmetic appearance, so that if necessary other conjunctival sutures should be applied (Fig. 16).

If necessary, the opening in the conjunctiva made in tenotomizing the antagonist should be closed with a suture. If an overeffect is feared, as shown by limitation of motion, a regulating stitch may be passed through the tendon of the tenotomized muscle and through its insertion, and too free a tenotomy regulated.

Time would fail to enumerate the various alternative methods proposed and the reasons for their rejection. In some cases the choice is not easy, since the superiority of the method I have selected or devised is not sufficiently great. Thus, the Worth method makes fast to the muscle by a suture which is tied in a firm knot outside the conjunctiva. This is a well tried, reliable

method. It is rejected in favor of the whip stitch, because the latter is simpler, is entirely secure and does not include the conjunctiva.

The methods which simply pass the suture through the muscle once on each side with no knot and no whip stitch are not reliable,

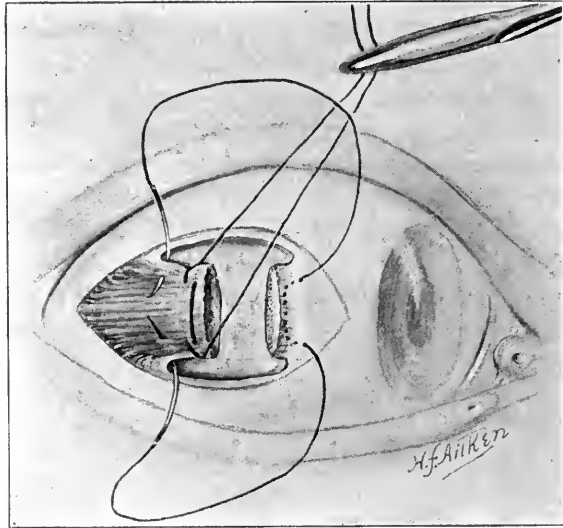


Fig. 14.

since the stitch so easily cuts its way along between the muscle fibers. If no strain is put on this kind of a stitch until it is tied down on the sclera or stump of tendon, as on Jackson's, Verhoeff's, Reese's and others, the hold is fairly secure but has been known to slip—no doubt, due to faulty technic in tying the knot. This form of suture is added to the primary suture as a security suture in the operation here described. Virtually the same thing has been recommended by Butler of England.

One great disadvantage of the tying-down suture of Reese, Verhoeff, Jackson, etc., is that there is virtually no latitude of adjustment in tying the suture—only in placing it. One must decide just how far back to place the suture in the muscle, and then tie the muscle down with that point closely applied to the sclera where the stitch enters that tissue. It is secure only when drawn firmly and so no adjustment is available. Sometimes a little adjustment is very desirable, but I do not wish to imply that one can expect to adjust the position of the eye during the operation even under local anesthesia, far less under narcosis, and expect the same balance to obtain a few days later. The

position depends on too delicate a nervous adjustment. This is put out of gear by the traumatism of the operation and the anesthesia.

The method of scleral anchorage advised is not the only good one, of course. It has these advantages: (1) It is most easy to apply because the eye can be held so firmly by the tendon stump, provided the direction of the stitch is away from the tendon towards the cornea, and (2) since the two stitches are tied in one transverse knot, the pull is divided between them and we have as strong a hold, tested by Bishop Harmon's post card method, as in the transverse stitch with the knot longitudinal.

An important consideration is the fact that so little strain is put on the tissue when the suture is being pulled up firmly in tying, since the direction of the pull by the surgeon is along the line of the thread toward the cornea, whereas when the thread passes transversely, as in many operations, the pull has to be deflected as by a pulley.

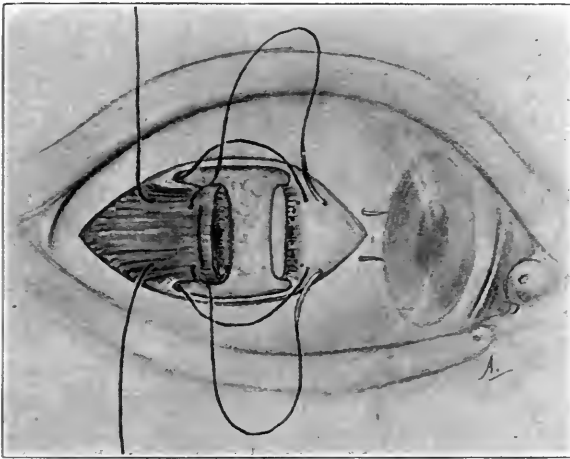


Fig. 15.

The two longitudinal stitches through the sclera afford a reliable hold even without the security stitch, provided they are well placed and well tied. The latter is added to provide for the occasional imperfectly applied stitch and to make the attachment so secure that a minimum period of bandaging will be required. It also makes unnecessary the curetting of the surfaces of muscles and sclera or their cauterization as proposed by Maddox. I do not approve of the French and German plan, following Landolt, of keeping the patient in bed with a binocular

bandage for a week, avoiding even a look at the eyes for fear the stitches will not hold. On the other hand, I prefer at least twenty-four hours in bed and a day or two more with both eyes closed, the operated eye being covered for a week or ten days. The conjunctival stitch may be removed on the second or third day. The security stitches may be removed on the fifth or sixth day, or left until the primary stitch is removed on the eighth or tenth day.

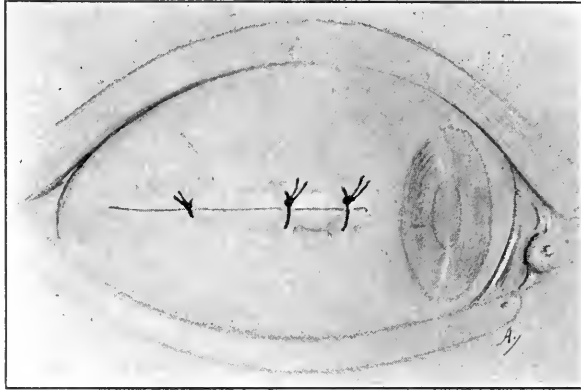


Fig. 16.

REASONS WHY THIS METHOD IS AS SATISFACTORY AS ANY
HERETOFORE OFFERED

1. Perfect anesthesia.
2. Hold on muscle is doubly secure.
3. Hold on the sclera is doubly secure.
4. The scleral stitch is inserted in the easiest way.
5. The primary stitch is adjustable, can be pulled tighter as trial shows necessary.
6. No special instruments required.
7. The security stitch secures close and effective apposition of muscle and sclera.
8. The security stitch stimulates plastic activity at the point where union of muscle and sclera is to take place.
9. The conjunctival incision closes with the best cosmetic result.
10. No buried stitches and no unsightly folds or lumps to mar the cosmetic result during the early weeks of convalescence, when the patient's attention and that of his friends is most closely concentrated on the eye.

Many operators, especially those with large experience, have already adopted some method of doing an advancement resection or tucking which is satisfactory to them. It is not to be expected that they will abandon a well tried and satisfactory method for a new one, however much is claimed for it. Those who have not yet found a satisfactory method, and they number not a few, are urged to give this a trial, comparing it with other methods, and see if the result is not satisfactory.

DISCUSSION

DR. EDWARD JACKSON, Denver: I agree with Dr. Lancaster as to the importance of these points that he has brought out in connection with muscle shortening or advancement; and, in the main, with his method of correcting such defects. When you are going to tie a thing down there can be no question as to which end of your rope should be fast. The thing would not be tied unless both are thoroughly fast, and this primary principle for every operation has been ignored and violated in scores of these operations. The result of operation of advancement, as done in the earlier years, was almost completely a matter of chance. No satisfactory operation for advancement can be done without both prompt healing of the muscle and firm hold on the tissues.

Many successful stitches have been devised. But most of these stitches get the hold by what Dr. Lancaster has called "puckering" on the tendon. You cannot get the hold on the parallel fibers of the tendon without something of a cross stitch. Any stitch between the fibers usually cuts out, and is wholly unreliable. If you try simply to tie across the tendon, you will always get a puckering up. The only way you can get a firm hold across without puckering is to fasten the tendon down on a firm base. You can thus stitch as tightly as you please and it holds the tendon spread out. There is the advantage in the "security stitch." It is the essential stitch and the one that holds the tendon firmly. You could put in half a dozen stitches that would be of no good if the other got loose.

There is a certain amount of risk in putting in one stitch and trusting wholly to it. But if the eyeball is turned toward the muscle to be advanced, and the tendon to be advanced is brought well forward and laid directly on the sclera, you can make a stitch without the risk of cutting through, and without puckering of the tendon. That is the advantage that appeals to me in the operation I have done for many years, and the essential thing in the Verhoeff operation and the one Darier advised ten years ago. The placing of this tangentially is more difficult than radially to the cornea, but not very much more so, if the angle is carefully chosen; I believe in putting your eggs in one basket and watching that basket.

With reference to the radial conjunctival incision, I do not know that the point was brought out strongly enough, that this kind of cut shows no tendency to gap afterward. Certainly this operation advised is a distinct advance over many given in the textbooks.

DR. H. B. LEMERE, Omaha, Neb.: I think this subject particularly timely on account of the qualifications required of the candidates for aviation. The requirements in regard to muscle balance are that exophoria or esophoria shall not exceed 2 degrees. If the disqualifying imbalance can be corrected and maintained, the candidate is accepted.

In curing this imbalance, it has been our custom to use the old Falk tucking operation; with this we have had very good success. We find this tuck excellent for the phorias and use it in both phorias and strabismus. I think the horizontal conjunctival incision employed by Major Lancaster should give a good operating field. We have found the choice of the needle to be very important. A full curved needle with a rounded noncutting shank will not cut out either through the muscle or at the anchorage. We feel that this round needle is more essential than any one thing for the security of the holding of the tuck. A tuck made with a mattress stitch and made with one of these rounding shank needles, will hold without any reinforcing loops. It will not cut through and it is not necessary to take such a deep hold in the sclera for anchorage, one firm insertion of the needle being enough to hold securely. We have found the vertical insertion of the anchorage more accurate and more efficient than the horizontal, and we are able to regulate the degrees of effect by this suture, testing the balance with prisms during the operation. For the suture we use the 10 day chromic catgut.

MAJOR ALLEN GREENWOOD: I call attention to a method of advancement which is now the principal one I show my students, because it gives everything desired and with no chance of a slip. A simple tuck is made and fastened, and then, instead of putting this fold or tuck backward, as originally advocated, it is fastened down tight by a suture through each corner of the tuck and the sclera, close up to the cornea. This gives an advancement of the muscle close to the cornea at the same time that a shortening is produced. It will not slip and I have never seen it fail. You can measure the amount of tucking according to the effect you wish to produce. It can be done quickly under local anesthesia. You can get as much effect as from a Worth advancement, with very much less traumatism. It is a simple method of advancement and tucking, which has appealed to my students and been adopted by them with good results. It can be performed by using the tenotomy hook in place of the tucker by lifting up a fold of muscle with the hook and pinching the fold together underneath with a pair of Prince's forceps and passing the sutures that hold the tuck beneath the blades of the forceps. I have found, however, that the Clark tucker is rather the better instrument for this particular combined operation.

DR. D. W. WELLS, Boston: An advancement that sutures the shortened tendon to the sclera at the original insertion is practically a resection and not an advancement; the structures have not been carried forward.

I have found one form of operation so perfectly satisfactory that I continue to use it. This is the Worth with the modification of the tangential scleral anchor. The important thing is to get it deep enough and in order to pass the needle through the tough sclera, one must have a sufficient counterpressure. To secure this I devised a fixation fork, with two very sharp points, the web being ground down to a knife edge. This is inserted through the conjunctiva into the sclera just opposite the point of inserting the needle the point of which emerges between the tines of the fork. Two or 3 mm. of sclera are included, and the depth should be two-thirds the thickness of the sclera. (I pass around a cut to show the ideal depth.)

Occasionally one scleral suture has cut out because not deep enough, but it has never caused any perceptible vertical deviation. I contend that a transverse suture gives a stronger hold.

DR. JAMES M. PATTON, Omaha, Neb.: I wish to ask as to the suture material he uses and the method of tying the suture.

DR. LUTHER C. PETER, Philadelphia: There are just two points which I wish to emphasize. (1) The success of any advancement operation depends largely on the work of the assistant. The eyeball should be rotated toward the muscle by the assistant. This can be done by a pair of forceps, or by Wells' fork. The muscle and eyeball should not be drawn together by means of the sutures. (2) The success of any advancement operation depends on keeping both eyes closed by means of a bandage for at least five days after the operation.

DR. C. W. HAWLEY, Chicago: I would like to enlarge on Dr. Greenwood's suggestion, that I have been doing it for five years, and to my knowledge, so far as the West is concerned, it is a modification brought out by Woodruff of Joliet.

First, inject, as Dr. Lancaster does, about 10 mg. of a 1 per cent. cocain solution with two or three drops of adrenalin in it. Make a vertical incision and instead of dissecting up the conjunctival and subconjunctival tissue, the conjunctiva only; then find the edge of the tendon and follow back this distance (illustrating) and make the Worth stitch here (illustrating). I have a special hook made like a strabismus hook and pick up the muscle and carry the upper needle back through the muscle as illustrated, carry the lower needle through the tendon at insertion, and then with this hook my assistant holds the muscle up and you bring the two portions together and simply tie them there. It is very simple and easy and very effective. The suture material is pyoktanin catgut. It has been demonstrated there is no union of tissue from the tendon on the eyeball between the tendon insertion and the cornea. You get no advancement of the tendon beyond the original insertion. To assist me in keeping the conjunctiva back, I take an ordinary lid retractor and find it better than the strabismus hook. Lately I have taken the smallest one I can get and have it ground off to 9 mm. in width.

MAJOR W. B. LANCASTER (closing discussion): In regard to the tangential hold, you remember that Bishop Harmon made some experiments on stitches passed through postal cards in different ways, from which he drew the obvious conclusion that a stitch so placed as to divide the pull equally between the two stitch holes, as would be the case when the stitch was inserted across the line of pull, would require more force to tear it out than a stitch of the same sized thread inserted parallel to the line of pull so that nearly all the strain would come on the more distant hole. This is the basis of Dr. Wells' criticism. It does not apply to my operation because the thread is double armed and each end is passed through the sclera parallel to the line of pull so that when the ends are tied we have the loop containing the knot transverse to the line of pull and so most favorably placed to divide the strain between the two holes. As a matter of fact the muscle is not strong enough to pull out the stitch when it is properly inserted in either direction. The all-important thing is to include in the stitch some of the tough fibers of the sclera. If this is done the stitch may be trusted to hold whether it is tangential or parallel to the line of pull. The method I suggest gives double safety because both needles have to be passed through the superficial layers of the sclera so that failure to place one would not spell disaster.

I use silk rather than catgut, which is too clumsy. I prefer a needle without an eye such as those used by some surgeons in suturing intestines and blood vessels. The thread comes out of the end of the needle which was flattened out and folded over the thread when it was made. Thus the thread is not double at the end of the needle, as it is when a needle with an eye is used. Such a needle and thread can be used only

once. It cannot be rethreaded. The advantage is that a sufficiently strong thread can be used with a fine needle and yet no difficulty be experienced in drawing the thread through the small hole made in the sclera by a fine needle.

Another advantage appears when you tie the suture. As the two ends of the thread are drawn on to pull the muscle forward, or to take up the slack, if it is pulled forward with forceps by the assistant, the two threads are both drawn straight through the sclera in the line of the original puncture and not around a corner as is the case when the stitch is placed tangentially. This facilitates the tying of the stitch in an important way. Moreover when the stitch has been tied in a single knot, or half knot as it is sometimes called, it will hold better than a similar knot at one side. The effect can be noted by the cover test or even by the rod test and readjustment effected by making the stitch tighter or looser.

As to keeping the eye closed five days, it is interesting, but I have not now time to go into that. A writer recently in an English publication states that he does this as an outpatient operation, not bandaging the eyes even for one day. This can be done if you have a secure stitch. At the other extreme are Landolt and others who say keep the eyes covered five days with the patient on his back and do not even open the eye to look at it because it is a gamble whether the stitch will hold as they place it, and if you touch it it may pull out resulting in failure. That is not so with the modern operation.

FOCAL INFECTIONS IN RELATION TO INTRA- OCULAR OPERATIONS

JAMES M. PATTON, M.D.

OMAHA, NEB.

The lack of uniformity with which tissues react to operative trauma has been one of the great handicaps in modern surgery. In the preantiseptic days, infections, inflammations and non-healing were ascribed to various causes, many of which were farfetched. With the introduction of antisepsis and later asepsis, surgeons fondly hoped that postoperative infections in the clean cases at least, would be a thing of the past and we are truly grateful for the work that has been done. But in the field of ophthalmology, at least, there is much yet to be desired. In a group of intraocular operations, each done with equal care as to asepsis and technic, some will heal with promptness and freedom from reaction, while others, without any apparent cause, will be irritable, congested, and painful and finally heal with more or less evidence of secondary inflammatory products.

Again in cases of penetrating wounds of the globe of accidental origin, a very extensive lacerating wound, even though infected, may heal more quietly and with better vision than a much less extensive injury that, clinically at least, should offer a better prognosis.

The attempt to answer this question has occupied the attention of ophthalmic surgeons for years, and while progress is noticeable, the unexplained cases of secondary inflammations are all too numerous.

It is well within the memory of some present when a careful irrigation of the conjunctival sac and external cleansing of the lids and lashes was all the preparation thought necessary or possible for an intraocular operation. Then attention was called to infected canaliculi and tear sacs with benefit to the patient and satisfaction to the operator. Next the influence of intranasal and sinus infections was advocated and although this was at first ridiculed by some very eminent ophthalmologists, we have all found, to our sorrow, that diseased sinuses can cause serious ocular inflammations.

But in spite of strict attention to asepsis, tear sacs, accessory sinuses, and such general conditions as diabetes and arterio-

sclerosis, inflammations and infections continue to be reported and it was the research men who finally came to our assistance.

It is obviously impractical to apply experimental laboratory methods to our private patients nor do we wish to subject our dispensary cases to artificially induced inflammations, but by comparing the results of animal experiments in the laboratory with clinical evidence as seen in our daily practice, we can draw some very definite conclusions. And, while the objection may be raised that an iridocyclitis, which is clinically secondary to an infected alveolus, is quite different from otherwise unexplained inflammations secondary to an operative or accidental wound in the presence of a similar root abscess, and while we cannot prove conclusively that the inflammation would not have appeared if there had been no focus of infection, at the same time, it recalls the theorem of our high school days that "Things equal to the same thing are equal to each other." In other words, if infected tonsils can cause irritation in an eye whose resistance is lowered by embolism, vascular changes, or special susceptibility as suggested by Brown and Irons, it is quite evident that a similar condition can ensue if the reduction in resistance is traumatic.

LITERATURE

It is unnecessary to review either the experimental or clinical literature of focal infection and their relation to ocular inflammations, but as we must rest our case in so far as the operative picture is concerned, largely on the findings just referred to, it will not be out of place to review briefly some of the conclusions more generally accepted by the profession at large.

We are all familiar with the experiments of Rosenow and his conclusions with reference to the apparent affinity of certain strains of microorganisms for definite anatomic structures. He further accounts for the high percentage of involvement of the anterior portion of the eyeball (*Journal of Infectious Diseases*, Vol. 17, P. 403) on an anatomic basis "as here we find a graduation from an abundant to a very scanty blood supply and hence a graduation of the available oxygen, thus inviting localization and affording opportunity for the growth of bacteria, a circumstance which for the same reasons would make for lesions by circulating toxic substances no matter from what source."

Billings (*Focal Infections*, P. 105) is convinced that "strains of streptococci in foci of infection of the teeth, tonsils and sinuses have an unquestionable relation to iridocyclitis alone as well as when the eye infection is associated with other general diseases."

Irons (*Journal Am. Med. Assn.*, Vol. 66, P. 1840) believes

that toxemias develop in foci protected from the resisting forces of the host. From these foci the products are thrown into the general circulation at varying periods, attacking such organs as may, through affinity or anatomic structure, be especially susceptible.

Brown and Irons (Abst. Pract. Med. Series, 1917, P. 59) analyzed 100 cases of iritis and they emphasized the fact that many cases were apparently the combined result of more than one causative factor, again reminding us that we should not be content to stop in our search for causes when one is found that seems to account for the trouble (fifty-five per cent. of the cases came under this head.) Of the remaining 45 per cent., each could be traced to some definite infective process.

Suker (*Ophthalmic Record*, Vol. 26, P. 278) reported a case of optic neuritis which cleared promptly on the enucleation of definitely infected tonsils.

Osborne (*Journal Am. Med. Assn.*, Vol. 69, P. 1313) calls especial attention to infections lurking under improperly fitted dental crowns and bridges and quotes Shamlind to the effect "that a toothless mouth is to be preferred to one containing a single focus that endangers the health of the patient."

Hardy (*American Journal of Ophthalmology*, Vol. 34, P. 97) reviews the recent literature on ocular diseases of dental origin and concludes that "not only are the teeth *capable* of causing ocular troubles but that frequently they do so." Inasmuch as a great many people have bad teeth who never have ocular inflammations, he urges that we "consider the teeth in our list of possible etiologic factors but to refrain from making this possibility a hobby to be ridden to death." He also urges a close cooperation between the oculist and the dentist.

Fox (*Ophthalmic Year Book*, Vol. 13, P. 377) reports two cases of postoperative infection which clinically were secondary to pyorrhea. The infections were controlled by prompt correction of the dental defects.

Many additional references could be made to the literature from the experience of close observers but would only emphasize the opinions already quoted.

POSTOPERATIVE IRRITATION

My attention was first called to postoperative irritation some years ago by observing how liable we were to have trouble in operating on an eye in a patient where the fellow eye had been lost through a previous operation, often in the hands of oculists whom we know to be careful, scientific and skilful operators. Thinking that in some way the shrunken globe had something

to do with the secondary inflammation, it was our custom to enucleate the stump, then wait several months before operating on the eye. In spite of this we were often disappointed and I recall one patient in whom a perfectly smooth cataract extraction was followed by unexplained iridocyclitis and loss of the eye.

Our attention was called to the influence of focal infection shortly after this distressing incident and we promptly instituted a thorough preoperative examination of all patients requiring intraocular operations as well as those with penetrating wounds of accidental origin. This examination includes the usual exploration of the conjunctiva, tear sac, nose, teeth, sinuses and tonsils and if there is any question at all as to their condition, the teeth and sinuses are rayed. If there is any history of intestinal, renal, pelvic or venereal trouble, the patient is examined by a competent internist and any evidence of focal infection corrected. This, especially in the case of teeth, tonsils or sinuses, may mean a delay of several days or weeks but the results have been so gratifying that we have felt it well worth while.

Of course, we have no way of proving that any of the individual patients would have done badly if they had not had this preoperative attention. On the other hand, we have had several patients who had lost an eye in a previous operation in which we found an apical infection, bad tonsils or some other focus and when these were corrected, the patient was operated on without subsequent irritation. Again, they might have gotten along without irritation but the evidence is all the other way. I saw the patient previously mentioned a few months ago and, on examination, found him to have several badly infected teeth, but this discovery came too late to do him any good.

There is, of course, the element of added expense and occasionally a patient will object to what they consider an entirely useless waste of time and will even be so unkind as to insinuate that they are being exploited for the benefit of the laboratory man and internist. But an explanation will usually convince the average patient of the advantage of the examination and if they flatly refuse, as a patient did for us a few days ago, explain the situation carefully, insist that they take all the responsibility, think of the many cases that turned out well before we knew anything about focal infections, and go on with the operation.

CONCLUSIONS

From the foregoing it seems that we are justified in drawing the following conclusions:

1. Inasmuch as ocular inflammations do occur in the presence of and apparently secondary to certain foci of infection it is reasonable to conclude that a traumatism, whether operative or accidental, in the presence of a similar focus, could light up or be followed by an unusual degree of inflammation.

2. Although it is frequently the tendency on the part of all of us to overestimate the value of new and popular theories, the average patient will agree that an eye is of much greater value than teeth or tonsils, especially when definitely proved that they are diseased.

3. This being true, it is our duty as oculists to promptly remove or correct such foci of infection before subjecting our patients to operative procedures.

DISCUSSION

DR. J. J. HOLMES, Lincoln, Neb.: The title of Dr. Patton's paper is of itself insufficient to bring many of us face to face with the fact that we have not been considering focal infections from this exact angle and we are to be congratulated on having this phase of the subject so clearly presented. There is, I believe, no chance for argument on the facts presented here as the Doctor has very wisely shunned that "No Man's Land" that lies between the theories and work of Elschmig and Brown, whose statements must be accepted as not proved. Therefore, we can accept focal infection as a liability to the results of ophthalmic surgery without regard to the *modus operandi* of the infection and as the time is becoming short and a great wealth of material is still to be considered in the few hours left to us, I will pass the opportunity of discussion on to those whose larger clinical experience will profit us more than anything that I shall be able to say.

DR. ARNOLD KNAPP, New York: Dr. Knapp thought that this was a very important communication, as it brought up for discussion a subject in the diagnosis of which advance is being made. He wished to refer particularly to two cases of cataract extraction which, without any cause in the operation, developed a severe iridocyclitis at the end of the first week after operation which did not respond to the usual treatment. On examining the teeth, abscessed roots were found in the upper jaw corresponding to the side of the operated-on eye. After removal of these teeth, in which an abscess bag was found adherent to a carious apex, the eye condition began to improve and recovered.

In both of these cases the type of iridocyclitis was a peculiar one. In addition to being severe, it was characterized by a hemorrhagic tendency. There was a hypopyon like exudate which was distinctly bloodstained and there were a number of nodules visible in the iris with punctate hemorrhages. It is this tendency to hemorrhages which he wants particularly to emphasize in this form of iridocyclitis.

DR. PATTON (closing discussion): In reply to Dr. Knapp, it is impossible to give this attention to all patients. Some will refuse to have it, but if patients have sufficient means to pay for the operation they also have sufficient to pay for the necessary laboratory preoperative tests. Those in the dispensary can be attended to in the dispensary laboratory and secure a sufficiently good examination for us to come to some conclusion.

DEFECTS IN EDUCATION FOR OPHTHALMIC PRACTICE

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The common and glaring defects in training for ophthalmic practice have arisen from imperfect preliminary and undergraduate medical training; and from the general failure to recognize that ophthalmology includes a mass of special facts and processes that will only be acquired through the personal relation of teacher and student.

At a time when every medical student was expected to learn how to use the stethoscope and clinical thermometer, not one in fifty was given instruction in the use of the ophthalmoscope. The preliminary requirements for entering the medical school took no account of parts of physics and mathematics fundamental to ophthalmology. When ophthalmology was recognized in the curriculum of undergraduate medical study, it was as a clinical branch given near the close of the course. It is still without proper recognition of the laboratory work and systematic training, on which alone the sound clinical teaching of it could be founded. Only in the last few years, and still in a minority of schools, have these defects been remedied.

The medical education of the student who has just received his doctor's degree is generally in an embryonic state; but a large proportion of those now engaged in the practice of medicine in this country have a medical education especially defective in respect to ophthalmology, and there has been little opportunity or encouragement to remedy these defects. The postgraduate schools, started a third of a century ago, were wholly clinical. Profitable clinical teaching presupposes a preliminary course of systematic study. The measurement and correction of astigmatism can only be intelligently carried out by one who knows what astigmatism is, and after its relation to emmetropia, hypermetropia, myopia, and accommodation are understood. The clinical characteristics of a corneal inflammation become significant in proportion as the observer knows the anatomy and pathologic tendencies of the cornea; and its relation to sclera, conjunctiva, lids, and iris.

The average graduate in medicine lacked the preparatory training that would enable him to profit by the clinical study of ophthalmology. If his teacher had not the same defects, to a degree that made him unconscious of them in the student, he was likely to become discouraged with the poor material he had to work on; and the misguided would-be specialist spent his six weeks in watching a parade of cases that he never fully understood or could profit by. Teaching so given is necessarily ineffective; and no repetition of such clinical courses could ever give a satisfactory education in ophthalmology. It is still too much the accepted model for ophthalmic teaching.

There has been a great improvement in some schools that give graduate teaching in ophthalmology. Systematic courses are now given extending from over three months to a year. And some instruction is given in ocular anatomy, pathology, and optics, along with the clinical work. But the best courses of this kind leave much to be desired; they are not given at all schools; and they are taken by only a minority of those who are preparing for ophthalmic practice. There seems to be no general understanding of the importance of these branches. A few private instructors have stimulated their assistants to do the needful study of these fundamentals. But too often there has been no suggestion to the student that anything more than attendance on a clinic was needed to make him an ophthalmologist.

It has been supposed in the past that an internship in an ophthalmic hospital was a superior opportunity for preparation for ophthalmic practice, and in many respects it is. But, on a young graduate in medicine, ignorant of ocular anatomy, pathology, and physiologic optics, such an opportunity is largely thrown away. A year or eighteen months' service in such a position may still leave him defective in the fundamental knowledge that would have made his internship truly valuable; and leave him unprepared to meet the needs of the cases that will form three-fourths of his work in private practice.

This is not a merely theoretic indictment of our present methods of teaching ophthalmology in this country. Every point in it has been supported and emphasized by a year's experience in the work of the American Board for Ophthalmic Examinations. These educational shortcomings appear in the case histories submitted, in the papers produced in written examinations, and in the laboratory and clinical work of the candidates. It is too much to suppose that they will not impair the quality of the service an ophthalmologist so poorly trained can render to

the community. I do not forget that every ophthalmologist has done something to remedy such defects, some attaining great success, others with but indifferent results. But not one of us is, or will be free from the defects entailed by lack of proper training. It is our business to see that those who come after us will have a better chance.

There is a proper sequence for the building of a house, or the learning to walk. There is an orderly effective way of bringing together the knowledge we have to use in ophthalmic practice; and learning to manipulate its instruments and apply its facts. Any one who has worked in ophthalmology can give some hints as to what this order and these facts and instruments are. We who have gone over this ground, however slowly, laboriously, and by devious ways, can point out to others some of the more direct and easier paths by which they can travel.

PRELIMINARY TRAINING

The preliminary studies of especial importance, apart from those preparatory to the study of medicine in general, are mathematics, optics, and drawing. Of mathematics, geometry and plane trigonometry are essential to an intelligent study of optics. So far as I know, plane trigonometry is nowhere required as a preliminary to the study of medicine. Yet it is hardly possible for one who does not know something of plane trigonometry to understand the first law of refraction, as it is commonly stated.

It is too much to expect that all medical students shall have trigonometry before entering on the study of physiologic optics. But it is not too much to expect that every physician who proposes to prepare himself adequately for ophthalmic practice shall study plane trigonometry. For such it is time saved. We have required it for the graduate degree in the University of Colorado. The first graduate student to take our course began by hunting up a high school teacher of mathematics, and taking under him a course in trigonometry. This plan is open to any one who lives within reach of a good high school, and the branch is one that can be mastered by a good student from the book alone.

Of algebra not much is required in preparation for an understanding study of ophthalmology; and this is furnished in most college or high school courses that require some mathematics. For advanced study, "descriptive" geometry and mathematical drawing will more profitably employ the student's time and energy. It is not too much to ask that any doctor of medi-

cine who expects to take up ophthalmic practice as his specialty, should begin by making good the mathematical foundation necessary for a good and early mastery of physiologic optics.

The needed study of optics is possible in any well-equipped university or advanced school of physical science. But the opportunities so offered are not particularly attractive or well suited to the student of ophthalmology. Physiologic optics is a special branch of the science, of interest chiefly to those who have studied medicine. The course arranged by the professor of physics does not go very far in that particular direction, and does give more of other parts of optical science than is needed by the ophthalmologist.

In a way, physiologic optics can be studied by the help of books. But it can only be mastered by carefully performed and thoroughly understood experiments. The essential laboratory fittings are neither elaborate nor expensive, but it should be largely a laboratory course. Special instruction in ophthalmology should begin with such a course. It might be arranged in connection with any graduate school of clinical medicine. But in fact a good course of the kind scarcely exists in America. Recently the best advice that could be given to a graduate student of large clinical experience, who wished to get a mastery of physiologic optics was that he should get Burch's "Practical Exercises in Physiologic Optics," and try to teach himself and a fellow student.

Drawing should have an important place in systematic training for ophthalmic practice. It may not be the only way, but it is an excellent way to get training in the accurate use of eye and hand that is greatly needed in ophthalmic work; and it is the only study usually given in an academic curriculum that does furnish this. It may be taken as preliminary to the study of medicine, in which case its value will be evident at all stages of the medical course. Or, it is worth while to cultivate it on taking up the special study of ophthalmology. Not much can be expected in the way of teaching it in graduate medical schools. But the student who has had its value pointed out, can work at it alone, or can find help from professional draftsmen or artists.

THE COURSE IN OPHTHALMOLOGY

The special training for ophthalmic practice needs to be greatly improved in these three directions; physiologic optics, anatomy, including the minute anatomy, and the pathology of

the eye. When the training in these branches has been made more thorough, the present facilities for clinical instruction will become more valuable and effective. Enough has already been said regarding physiologic optics in connection with preliminary training. But for the near future we will have to depend largely on graduate schools in ophthalmology to furnish it. They should take it up with the purpose of making it thorough, and of giving it before much time is given to clinical work.

The anatomy of the eye and of closely related parts need not claim much time in the graduate course. Some things about it are fairly well taught in the general medical course. The most serious criticism of such teaching is that it does not go sufficiently into minutiae to meet the needs of special practice; and it does not deal sufficiently with the fresh or living eyeball. If anatomic material of the proper kind is close at hand, this instruction can be given in connection with operative or other clinical work. The essential thing is to emphasize the important anatomic facts, and not to allow them to be overlooked in the concentration of attention on operative or therapeutic procedures.

For the proper teaching of ocular pathology a course must largely be built up *de novo*. Not that good courses in pathology have not been offered from time to time in the past, for they have, but they have not often been taken or given. For students trained as good medical schools now train their undergraduates in general pathology, the course in ocular pathology need not be a long one. Perhaps one month of well planned, intensive training would be fairly effective. But it is a part of the course that needs to be carried out under the eye of a master. In few branches is the timely hint, or the correction of faulty technic and inference, so important. It is not merely a matter of learning to recognize certain tumors and pathogenic bacteria, it is to learn to see and appreciate all ordinary pathologic changes in all the ocular tissues. The course in ocular pathology will more and more be the test of the thoroughness of the teaching of ophthalmology in any special graduate school.

The clinical work is the part of the teaching of ophthalmology that is now best developed, and can best be trusted to take care of itself in the immediate future. When it is sought by students well prepared in the branches already alluded to, it will inevitably become more effective. But a little appreciation of the general principles of pedagogy would greatly improve it. Between the uninspiring narration or pumping in of facts, and the abandonment of the student to his own powers of observation

and deduction, lies the whole realm of teaching. There seems to be a very strong tendency to abandon this realm for the easy routine on either side. It requires more exertion to find out what the student knows or understands, and connect this with what he needs to learn, than it does to prose forth the line of thought that passes through the teacher's mind. And, it is still easier to bring the student before a patient and abandon him to his own devices. Perpetual stirring up is required to keep clinical teaching effective and thorough.

In this department of clinical work the most important portions are diagnosis, and exact estimation of errors of refraction. The examinations of the American Board show that while every one who comes up has tried to gain some skill in these directions, the general lack of thoroughness and skilled guidance is painfully evident. Men of rather large clinical experience have fallen down woefully in regard to the recognition of intraocular conditions, or the exact estimation of refractive errors. One who has passed a full internship in an ophthalmic hospital may be quite ignorant of any systematic method of hunting down an error of refraction; and show lack of any instruction as to the systematic use of the ophthalmoscope.

Case histories have shown strikingly deficient training in their taking and recording, even among those who have had years of clinical and hospital experience. In this respect ophthalmologists compare but poorly with the better trained of general surgeons (American College Surgeons). There are reasons for which the surgeon or the neurologist should be stimulated to keep complete records of his cases that do not apply with equal force to the ophthalmologist. But making all due allowances, the deficiencies in the matter of case histories that have been brought to light by the experience of examiners in the American Board for Ophthalmic Examinations are very serious. The attention of all teachers of ophthalmology should be called to them, and the need for better work in this direction emphasized by the publication of the statistics.

REMEDIES

The ophthalmic training of every student of medicine should include some careful instruction with regard to case taking and case records. This should be made a center of organization for his knowledge of ophthalmic diagnosis, the branch of ophthalmology most important for the medical man who does not enter especially into ophthalmic practice.

The worst defects in present training for ophthalmic practice would be met if each university that has a medical department would establish short courses in physiologic optics, ocular anatomy, and ocular pathology, and would bring these to the notice of every medical student, as courses to be taken before seeking the clinical part of training for ophthalmic practice. Even though he manifests no immediate interest in them, he would think of them when his attention began to turn to ophthalmology as a specialty. If the whole medical profession understood the importance of such instruction, it could better judge the qualifications of the men who claim to be specialists in this direction.

Such courses should be established in connection with each graduate medical school that undertakes to teach ophthalmology, and the would-be student urged to devote the first weeks of his course chiefly or entirely to them. Every leader or teacher in ophthalmology has a duty to the profession and to the public in this matter; and the announcement of the medical school can be made a most important instrument to bring about reform in this direction.

Those of us who are now engaged in ophthalmic practice are more or less conscious of our own deficiencies in regard to these studies fundamental to ophthalmology. It will be of great benefit to us individually as practitioners, to do something to remedy these deficiencies; and an open concerted effort to do so will do more than anything else to enforce the importance of fundamental training on those who intend to make themselves ophthalmologists.

An organization like this could arrange to give, just before its annual meeting, a week of intensive training on the microscopic diagnosis of intraocular tumors, under three or four of its members who have given special attention to ocular pathology. Or, to make the best use of the time with the least strain of any one set of powers, this might be combined with a similar course on experimental optics, or the minute anatomy of the eye with reference to operations on the anterior segment, or the location of foreign bodies. Such courses should be put on a basis of fees that would compensate the instructors, and provide the necessary material or equipment. Similar courses could be arranged by local ophthalmologic societies for the benefit of their own members, and to suit their convenience.

Finally, it would help to remove the greatest defects in training for ophthalmic practice; it would emphasize the importance of fundamental training, if the American Board for Oph-

thalmic Examinations would divide its examinations into two parts. It could give to students who have been out of the medical school one year or more, an examination on ocular anatomy, the physiology of vision, geometric, experimental, and physiologic optics, and in ocular pathology. Two or more years later it could give the examination in ophthalmoscopy, and other branches of clinical diagnosis, clinical examination of patients, therapeutics, and operative technic. Something of this kind is needed to prevent the essential preliminary work from being neglected; and to prevent such neglect from impairing the value of all training in the so-called practical branches.

INDEMNITIES FOLLOWING INJURIES TO EYES HOW SHALL WE DETERMINE THEM?

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My apology for presenting a paper of so little scientific nature is the fact that it deals with a problem that has confronted and will confront probably every ophthalmologist in this country; a problem that has caused much discussion and argument in many cases of industrial eye injuries when the question of indemnity was up for determination.

In these days of military activity we must not forget that we have industrial responsibilities. The purpose of this paper is to deal with one of these responsibilities of the ophthalmologist industrially, although the points for discussion will no doubt play a very important part in military ophthalmology before the government bureaus after the close of the war.

The determination of the amount of vision remaining and the percentage amount of vision lost, following injuries to one or both eyes, may seem to many of you a very simple matter. I am confident, however, that the majority of the members find this matter a real problem. It is also well to speak of the percentage of loss of efficiency suffered by a person on account of injury to one or both eyes. These are really very different problems which at first thought might be considered identical.

Dr. E. E. Holt of Portland, Me., has worked out an ultra-scientific method of computing the loss of efficiency due to loss of different portions of the field of vision in one or both eyes as well as efficiency loss due to reduction in acuity of central vision, and also that due to loss of use of one or several of the muscles of one or both eyes.

DETERMINATION OF INDEMNITY

Every case presents individual points for consideration and must be decided on its own peculiar merits. Loss of efficiency on account of loss of vision caused by occupational diseases (distinctly not accidents) affecting the eyes, is a matter that requires special study. The results following some injuries may cause considerable loss of general efficiency in a person and not particularly impair his efficiency in some certain line of work.

Loss of one eye would seriously affect stereoscopic vision; yet a watchmaker or a master of fine engraving does excellent work with a single eye and glass.

Some state industrial accident commissions rate the loss of efficiency by the person's ability in actual earning power after injury as compared to the earning power before injury.

An eye may have enough vision remaining to enable its owner to do certain kinds of work, but not enough to enable him to do the kind of work he was doing before the injury.

It does not necessarily follow that the man who has lost an eye is totally and permanently reduced in efficiency and earning power. He may turn to a different vocation in which by diligent application he may so cultivate his faculties that his earning power will be greater than before. Pennsylvania recognizes this and solves the problem by providing that:

"For the loss of an eye an employe is entitled to compensation for 125 weeks at the rate of 50 per cent. of his wages. For partial loss of an eye he is entitled to only 50 per cent. of his wages during total disability. If, however, upon returning to his work he is unable to earn as much as he did previous to his accident his employer must pay him 50 per cent. of the difference in his earning power for a period not exceeding 300 weeks or until his earning power has increased in amount equal to that previous to the injury."

Facial disfigurement may add to a person's loss of efficiency in the competitions of life. Is an eye of unsightly appearance a facial disfigurement? If so there must be a wide range of ratings on such. From merely the deviation of an amblyopic eye, the globe of which is of normal appearance, through the various cosmetic results caused by a chronic congestive conjunctivitis; the white pupil of a traumatic cataract; epiphora following destruction of lachrymal canals; coloboma of lids; enucleation of globe, with or without prothesis, to total destruction of the globe and orbit. Again, what position should we take regarding facial disfigurement when an artificial eye is worn following enucleation? Clearly the wearing of a well fitting artificial eye reduces disfigurement to a great degree but does not entirely eliminate it.

Many disfigurements of the eye and its appendages are amenable to plastic surgery. I have in mind a case wherein I was enabled to eliminate an old disfiguring coloboma of the upper eyelid by inlaying a piece of skin with hair from the eyebrow. Even living eyelashes were supplied. An eye may be blind without contributing to disfigurement. An eye or its

appendages may contribute largely to disfigurement without serious impairment of vision, for example, ectropion. Yet, if this ectropion persists the vision may be impaired as a consequence.

Damage to an eye or its appendages or vision may occur as the remote result of injuries to other structures, for example, paralysis or blindness may result from an injury to the skull or the brain, epiphora from an injury to the nose, and ectropion from the contraction of a scar following injury to the face.

INTERPRETATION OF LAWS ON THE SUBJECT

There is opportunity for much difference of opinion regarding statements in accident insurance policies, state industrial laws, and other laws, concerning damage to the eyes, on account of the different words and expressions used interchangeably which permit of different interpretations. These should be made more uniform with exact definitions. There is a possible chance for difference in the interpretation of what is meant by "vision" and by "sight." I would interpret vision as the ability of the eye to determine the relative size, shape and color of objects by means of sight. Sight being the power of recognizing light and thus distinguishing between light and darkness. Vision is lost when the relative size, shape and color of objects can no longer be determined by the eye. Sight is lost only when the eye can no longer determine between light and darkness—absolute loss of light perception. With the eyelids closed over the eyes the normal eye can distinguish between light and darkness and can perceive the hand passing between it and the light, but it cannot tell the relative size, shape or color of the hand. The sight remains; the vision is gone.

The position has been taken by accident insurance officials, concerning policies providing indemnity for total loss of an eye: "That the eye is not totally lost as long as there is any light perception remaining." Hence, benefits do not apply.

The following questions are frequently put to the ophthalmologist in court and in private settlement of claims following eye injuries and in interpreting the law: What is "Loss of an eye"; "Loss of vision"; "Loss of sight"; "Blindness"? Each in a degree total or partial; and if partial, to what percentage of total; or of normal.

The expressions, "vocationally blind," "industrially blind," and "totally blind" are subject to different interpretations. The only term describing the percentage of blindness which seems to

be universally accepted as to its meaning and admits of no equivocation is the expression "stone blind."

On the proper interpretation of these terms and conditions depends in a great measure the ability of industrial boards, accident insurance companies or other judicial bodies to administer with equal justice. The lack of uniformity is apparent when the industrial accident compensation laws of different states are studied.

Utah provides indemnity for loss of an eye and makes a distinction between loss of an eye "by enucleation" = 120; and "total blindness" = 100. Utah also considers total blindness of both eyes as a total and permanent disability; but makes no provision concerning facial disfigurement. Mr. P. A. Thatcher, Chairman of the Utah Industrial Commission, writes:

"There is nothing prescribed in our Act for the partial loss of vision * * * * * and up to the present time we have not had occasion to hear any case where the partial loss of vision has been a consideration."

Minnesota provides indemnity "for loss of an eye" and also provides that "the total and permanent loss of the sight of both eyes * * * * * shall constitute total disability."

Quoting from a letter statement by the Department of Labor and Industries of Minnesota: "The amount of compensation * * * * * depends upon the percentage of loss of vision which he (employee) has sustained."

I have been unable to get information as to how this vision percentage is determined by the Minnesota Board.

Pennsylvania provides compensation "for the loss of an eye." "The Workmen's Compensation Board has ruled on several occasions that an employee is not entitled to compensation for the loss of an eye unless the vision of same is entirely destroyed." Thus it is very important to definitely determine when vision is, or is not, "entirely destroyed."

Wisconsin provides indemnity:

"For loss of an eye by enucleation = 160. For loss of the second eye by enucleation = 320. For total blindness of one eye = 120. For total blindness of the second eye = 240."

Total blindness of both eyes is considered an equivalent to, and rated the same as, total disability. It does not specify just what is meant by "blindness" and here again is need for a positive definition of the word "blindness." Wisconsin also provides indemnity for facial disfigurements.

Connecticut provides that:

"Total and permanent loss of sight in both eyes, or the reduction to one-tenth or less of normal vision with glasses * * * * * shall be considered as causing total incapacity and compensation shall be paid accordingly."

Regarding injury to one eye provision is made:

"For the complete and permanent loss of sight in one eye or the reduction in one eye to one-tenth or less of normal vision with glasses."

There is no limit put on the diopter strength of lenses necessary to bring vision to as near normal as is possible. Therefore, single or double traumatic cataract could be operated on and correcting lenses worn and the vision would be increased to such an extent that the compensation covering traumatic cataract in Connecticut would be no more than half that in Oregon where traumatic cataract is considered as total loss of an eye.

Connecticut does not fix a specified amount payable in percentage of the whole according to percentage lost, in cases of partial loss of vision. It generalizes all injuries causing partial incapacity and, like Pennsylvania, provides the indemnity, "equal to half the difference between his average weekly earnings before the injury and the amount he is able to earn thereafter," apparently irrespective of the percentage of vision remaining; and also not limiting the earning capacity to the same kind of work done before the injury, but permits the employer to procure for the injured employee employment "suitable to his capacity."

Colorado provides indemnity for:

"Total loss of an eye by enucleation = 139; for total blindness of one eye = 104" with provision that "in all other cases * * * * * the compensation shall bear such relation to the amount stated * * * * * as the disabilities bear to those produced by the injuries named."

Colorado therefore does make a distinction between "loss of an eye" and "total blindness of one eye." Provision is made for indemnity covering serious permanent facial disfigurement.

Ohio provides indemnity "for the loss of an eye" at the same rating that it provides for permanent total disability limiting the period over which indemnity is to be paid. It does not specify what is meant by "loss of an eye."

Nevada specifies that "the total and permanent loss of sight of both eyes" shall be considered "total and permanent disability in absence of proof to the contrary." This state provides in-

demnity "for the loss of an eye" and explains that "the permanent and complete loss of sight in one eye may be deemed as the loss of one eye." And, "in all cases of permanent partial disability, not otherwise specified in the foregoing schedule, the percentage of disability to the total disability shall be determined." Nevada also makes a distinction with reference to

"A disability that is partial in character but permanent in quality. Where a percentage loss of vision is reported to the Commission by the eye specialist, to whom the claimant was referred, the compensation awarded is determined by multiplying the percentage loss of vision by the compensation for the total loss of vision. Loss of 90 per cent. of the vision of an eye is considered as total loss of the eye."

There is a provision as follows:

"Where there is a previous permanent disability, as the loss of one eye, * * * * * the percentage of disability for a subsequent injury shall be determined by computing the percentage of the entire disability and deducting therefrom the percentage of the previous disability as it existed at the time of the subsequent injury."

Nevada provides a further indemnity under the heading of "Facial disfigurement. For permanent disfigurement about the head or face which shall include injury to or loss of teeth."

Montana provides indemnity for the loss of "one eye by enucleation = 120"; and for "total blindness of one eye = 100." There is provision made for compensation for injury producing "temporary total disability" and, "for an injury producing total disability permanent in character" and, "for an injury producing partial disability." Presumably these provisions apply to injuries to eyes as well as to other parts of the body. I find nothing specified regarding facial disfigurements.

Oregon considers "total loss of eyesight" as permanent total disability, and "loss of one eye" as permanent partial disability.

Later in the same section of the law reference is made to this permanent total disability as "the permanent and complete loss of the sight of one eye." On inquiry I learn that in Oregon traumatic cataract is considered total loss of vision and the same award made as for total loss of the eyeball. This is unfair. A man with traumatic cataract has not suffered nearly as much loss as has one with globe entirely destroyed. The former is not a total, permanent, irretrievable loss as is the latter.

The following letter recived in reply to mine is interesting:

"Replying to your letter of recent date to the State Industrial

Accident Commission relative to the method of determining in terms of percentage the loss of vision a workman may have suffered, I will say that when an injured workman has lost some vision as a result of accident, the case is allowed to remain for three or four months after treatment has ceased in order that one may be sure that there will be no further improvement or further loss of vision and then the eye is tested for distant vision taking 20/20 as a standard, or equal to 100 per cent. If the workman's vision should be found to be only 10/20, it would be considered that he had lost one-half of the vision in that eye, etc.

When there is a loss of useful vision and only perception of light remains, the vision of the eye is considered as totally lost and award made accordingly.

In cases of traumatic cataract, the accident commission does not insist upon an operation to restore vision because that eye would be aphakic and would not therefore accommodate with the other eye, so in this case the injured workman would be paid for the loss of vision. The same award is made for total loss of the eyeball as is made for loss of vision.

I realize that sometimes these cases must be determined rather arbitrarily, as for instance in a case of contracture of the field vision, due to skull fracture where the perimenter record will show good central vision but very little vision outside of central vision, such a party might be able to do office work as well as before the accident but certainly could not go out into the woods and work at logging as he did before.

Then to state in general terms, normal vision considering it is 20/20, we rate as 100 per cent. and estimate the loss of vision by test of vision at 20 feet with the regular test type, converting the fraction into 100ths in order to make the estimation of percentage more convenient."

Oregon furnishes a very complete, well designed blank for eye surgeon's special reports on accident cases.

Wyoming provides indemnity "for loss of an eye." Provision for "temporary total disability" is made but "as soon as recovery is so complete that the earning power of the workman at any kind of work is restored the payments shall cease." "Total loss of eyesight" is considered permanent total disability:

"Where there has been a previous disability as the loss of one eye * * * * * the percentage of disability for a subsequent injury shall be determined by deducting therefrom the percentage of the previous disability as it existed at the time of the subsequent injury."

Michigan provides compensation for loss of an eye:

"The law in this State (Michigan) relative to eye cases was settled in the case of *Hurschkorn vs. The Fiege Desk Company*, Page 206, *Michigan's Workmen's Compensation Cases*, 1916. In

this case it was held that there had to be a total loss of vision, or over 90 per cent., before it would be considered loss of vision. No compensation is paid for partial loss of vision, other than what is paid for the time lost, except in cases where earning power of the employee is decreased. The loss of vision in both eyes is considered total disability. In cataract cases the Industrial Accident Board takes into consideration the fact that the eye upon which the operation is performed is only a reserve eye and will allow settlement on a partial loss of vision basis." A statement that is somewhat ambiguous.

Oklahoma under "Permanent Partial Disability * * * * * disability partial in character but permanent in quality" provides compensation "for the loss of an eye"; and states that "permanent loss of the use of * * * * * an eye shall be considered as the equivalent of the loss of such eye."

California has a very elaborate set of ratings and tables by reference to which, four items being first determined, the compensation to a workman for any particular kind of injury at any particular kind of employment can be accurately determined. These four items to be determined accurately as soon as possible after injury are:

1. The nature of the physical injury or disfigurement.
2. The occupation of the injured person.
3. The age of the injured person.
4. Average weekly wage of the injured person.

The list of results of eye injuries specifies as follows:

- "Complete loss of both eyes.
- Complete loss of the sight of both eyes.
- Complete loss of one eye.
- Complete loss of the sight of one eye.

Complete loss of the sight of one eye plus such impairment of the sight of the other as prevents reading or writing but not the ability to find one's way:

(a) Slight; (b) moderate; the degree to be determined by aid of medical advice, and (c) severe.

Loss of the sight of one eye leaving no scar or blemish such as would afford an observer evidence of such loss.

Permanent impairment of the vision of both eyes to the extent of rendering them useless for purposes of high visual requirement, but not for finding one's way: (a) Slight; (b) moderate, the degree to be determined by the aid of medical advice, and (c) severe.

Such a permanent impairment of the vision of one eye as renders it useless for purposes of high visual requirement, but not affecting one's ability to find one's way, the other eye being uninjured.

Hemorrhage of the eye, causing defective vision at times only.

Paralysis of the muscles of both eyes by reason of injury to the sockets causing double vision.

Paralysis of the muscles of one eye by reason of injury to the socket of that eye, causing immobility and double vision.

Injury to the eyesocket, causing immobility of eyeball with attendant impairment of range of vision only.

Laceration of lachrymal duct, causing chronic overflow of tears,"

and provides definite compensation according to the four cardinal items above stated for each one.

A recent change in the California Act provides that:

"The term 'injury' as used in this Act shall include any injury or disease arising out of the employment. In case of aggravation of any disease existing prior to such injury, compensation shall be allowed only for such portion of the disability due to the aggravation of such prior disease as may reasonably be attributed to the injury."

This provision makes it important that all employees pass through a physical examination before being put to work.

California provides under ratings for injuries to the face:

"Such injuries of the nose and face as by reason of the disfigurement, make the injured person so repulsive as to interfere with his ability to compete in obtaining employment, there being no permanent functional impairment," in three degrees of severity: Slight, moderate and severe, with a separate rating for each according to the four cardinal items.

The California act specifies that:

"Remediable injuries will not be considered permanent until all adequate and reasonable operations and treatments known to medical science have been used to correct the results."

"The law allows unlimited medical cost to meet this case," recognizing that "It is better to correct the trouble than to continue payments for permanent disability due to lack of attempts at correction."

EXAMINATION OF EMPLOYEES

A number of years ago I pointed out in an editorial in the *Journal of the Michigan State Medical Association* the necessity of examining the eyes of prospective employees before they are taken into employment. The wisdom of this course as a routine has been demonstrated many times. Some large employers of labor are doing it and I cannot understand why most are not. I believe there will come a time when a bureau will be established which will provide accurate information from records to all employers of labor and industrial commissions concerning the

physical condition of any one whom they are about to employ or about whom such informatin is needed. A clearing house by means of which the employer will not only receive protection in case of industrial accidents, but, knowing the physical condition of the man he is hiring, especially as to his vision and hearing, will be able to place the man at proper work to the mutual advantage of both employer and employee. Until such time when something of this sort is done, we are bound to accept the vision as normal before injury lacking proof to the contrary.

FINAL TESTS FOR VISION

It appears to be generally conceded that a reasonable period of time should elapse between recovery of an injured eye and the final testing of its remaining vision in order to learn whether the vision is going to become better or worse, as the case may be, so that the final vision test may be made with the eye at its probable mean level of efficiency. It is also permissible to use correcting lenses in determining the remaining vision. Some restriction should govern this, however, as pointed out in the report of the Chicago Ophthalmological Society's Committee, January, 1917.

This Committee, composed of Drs. Harry Gradle, Frank Brawley, and Harry Woodruff, recommended that there should not be a difference in spherical strength between the correcting lenses for the eyes of an individual to exceed 4 diopters.

Accepting as full vision the standard Snellen letter that subtends an angle of 5 minutes on the retina, normal vision may be represented by the fraction 20/20. Any increase in the denominator means a loss in vision certainly and any fraction thus formed represents a certain definite amount of vision. But what does it represent in percentage of full vision? This must be determined if we are to fix a definite percentage of indemnity due for partial loss of vision in an injured eye. Suppose that a man has vision of 20/40 with a correcting lens, in an eye at recovery from its injury. He had 20/20 vision before injury. Arithmetically speaking 20/40 is half of 20/20; *BUT it does not follow that the man has lost 50 per cent. of his vision.* It is impossible to establish a percentage without two fixed points; one high and one low. All points between these are definite percentages of one or the other as the case may be. 20/20 vision may be considered as 100 per cent. We must establish some definite amount of vision as 0.

WHAT CONSTITUTES TOTAL LOSS OF AN EYE?

Whether an eye must be actually lost in the sense that a finger is lost, that is, by separation from the body; or whether it is only necessary that vision be absent, the globe remaining, are the questions to be decided. If the latter, then must there

| Chapman's Percentage Vision Table. | | | | | | | | | |
|------------------------------------|---|------|---|--------|---|----------------|------------|--------|---|
| V A Chapman M.D. Milwaukee Wis. | | | | | | | | | |
| 20/15 | = | 100% | + | Vision | = | Superexcellent | Vision | | |
| 20/20 | = | 100% | | " | " | = | No loss of | Vision | |
| 20/30 | = | 95% | | " | " | = | 5% | " | " |
| 20/40 | = | 90% | | " | " | = | 10% | " | " |
| 20/50 | = | 85% | | " | " | = | 15% | " | " |
| 20/60 | = | 80% | | " | " | = | 20% | " | " |
| 20/70 | = | 75% | | " | " | = | 25% | " | " |
| 20/80 | = | 70% | | " | " | = | 30% | " | " |
| 20/90 | = | 65% | | " | " | = | 35% | " | " |
| 20/100 | = | 60% | | " | " | = | 40% | " | " |
| 20/110 | = | 55% | | " | " | = | 45% | " | " |
| 20/120 | = | 50% | | " | " | = | 50% | " | " |
| 20/130 | = | 45% | | " | " | = | 55% | " | " |
| 20/140 | = | 40% | | " | " | = | 60% | " | " |
| 20/150 | = | 35% | | " | " | = | 65% | " | " |
| 20/160 | = | 30% | | " | " | = | 70% | " | " |
| 20/170 | = | 25% | | " | " | = | 75% | " | " |
| 20/180 | = | 20% | | " | " | = | 80% | " | " |
| 20/190 | = | 15% | | " | " | = | 85% | " | " |
| 20/200 | = | 10% | | " | " | = | 90% | " | " |
| 20/210 | = | 5% | | " | " | = | 95% | " | " |
| 20/220 | = | 0 | | " | " | = | 100% | " | " |

Fig. 1

be complete loss of light perception before vision may be considered totally lost? If not, then how much remaining perception may an eye have and still be considered as having vision lost? This can only be fixed arbitrarily. I suggest that it be 20/220 vision.

There are probably certain areas or islands in the vision scale between 20/20 and 20/220 in which the vision so represented is of more value than others; and of different value in different persons according to the principal use to which the eye is put. Dr. Parker points out that:

"Failure of vision from just inside to just outside ability to read should have decided difference in percentage loss."

The personal value of remaining vision to a patient really increases very much as it approaches total blindness. This can only be fully appreciated by the "stone blind."

I suggest the preceding table as a working standard for determining vision in percentage (Fig. 1).

CHARTS AS AIDS IN DIAGNOSIS

I would suggest that charts covering this entire range of vision would be aids in arriving at a definite conclusion. If such

| CHAPMAN'S CHARTS | | | |
|------------------------------|----------------|------------------------------|----------------|
| PERCENTAGE VISION | | | |
| <small>100% 1000</small> | HTZAEN | <small>100% 1000</small> | ZETAHN |
| <small>80% 800</small> | NPEHT | <small>80% 800</small> | PHNET |
| <small>60% 600</small> | TAVZ | <small>60% 600</small> | VATH |
| <small>40% 400</small> | PHN | <small>40% 400</small> | HNP |
| <small>20% 200</small> | ZP | <small>20% 200</small> | EN |
| <small>10% 100</small> | PNH | <small>10% 100</small> | NPH |
| <small>5% 50</small> | VTAH | <small>5% 50</small> | ATVZ |
| <small>2% 20</small> | ENPHT | <small>2% 20</small> | HNETP |
| <small>1% 10</small> | THEAZN | <small>1% 10</small> | AZHETN |
| <small>0.5% 5</small> | HAZTNEV | <small>0.5% 5</small> | ZHTNEVA |

Fig. 2

charts were made I would suggest further that they be arranged with the largest types or devices in the center and the other lines placed above and below the center alternately, as they range in

the scale, so that the chart may be read up and down from the center. This arrangement gives the chart a certain value in detecting malingerers who insist on recognizing only a certain number of lines from the largest. Letters, numerals or characters may be used.

It would make a large unhandy chart if this whole table test were printed on one card. It has therefore been arranged on two semi-color cards; each with ten lines above and ten lines below the central letters as above (Fig. 2):

There is need for an illiterate chart in testing the vision of foreigners and uneducated laborers. I have never yet seen an

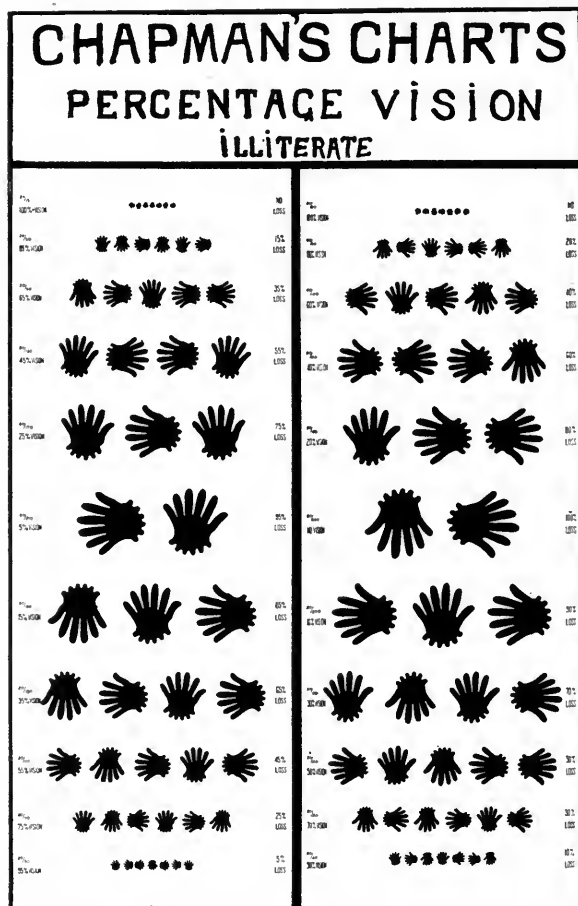


Fig. 3
1213 Wells Building

illiterate test chart that satisfied me and I have tried many during several years' work as examiner for the public schools of

the city of Muskegon. Not that they are particularly inaccurate, but because they require too much explanation in teaching the subject what he must do in order to use them. There is no letter, number, device or object with which the child or illiterate person is so familiar as the human hand; his own hand if you please, and I believe that a picture of the hand is the best device to place on an illiterate chart. The space between the thumb and first finger is too great in proportion to the space between the other fingers. I have therefore inserted an extra finger in that gap. The illiterate will not stop to count the fingers and only recognizes the picture as an open hand with the fingers pointing in a certain direction. It is very simple for him to hold his own hand up in a manner corresponding to the hand on the chart.

We then have the six fingered hand turned in different directions with a scalloped gauntlet cuff masking the proximal end of the hand in order to make it, as much as may be, no more readily distinguishable than the distal end. This device I have arranged in the same manner as the letters on the table charts: (Fig. 3).

DISCUSSION

DR. D. T. VAIL, Cincinnati: Dr. Harry Gradle of Chicago has published a practical scheme for determining the percentage of loss of vision on an economic basis for financial adjustment. It, like the essayist's, was ingenious and simple but not adaptable to all cases. I had the following case referred to me by the Ohio Industrial Commission for opinion and I wrote to Dr. Gradle for his opinion as his formula failed to meet the requirements of the case: A man, aged about 40, an industrial steel worker on "sky scrapers," was struck on the left parietal eminence by a 2 by 4 plank and as a result has complete hemianopsia. His vision at the center is 20/20 and he reads Ja. No. 1, but it is manifestly unsafe for him to climb about on skeleton iron frame work hundreds of feet high. According to the essayist's table he has 100 per cent. vision. Gradle replied his scheme failed to meet the demand of the case and estimated 30 per cent. of a year's salary I believe as being fair.

It is difficult to formulate a simple and efficient plan for estimating the percentages of loss in almost every case, nevertheless, we need something definite which can be understood by contractors, commissions, insurance companies, juries and others.

DR. V. A. CHAPMAN (closing discussion): In my paper I pointed out that every case has its individual points that must be considered, and must be decided upon its individual merits. I do not think it is possible to give a rule that will cover all cases. I also spoke of the loss of the field of vision. This table simply refers to central acuity of vision in terms of percentage. As it is now, we have no way to determine it. The question is put: "What percentage of vision does this man have?" I have never heard a definite answer. That was what I tried to get out of the Industrial Commissions. The only definite statement received was that they simply took 20/20 as 100 per cent. and called 20/40 one-half of that and said "there is so much loss in percentage."

I can not understand this position as commonly accepted regarding percentage vision and percentage vision loss. The fraction 20/20 simply means that the eye sees at 20 feet what a normal eye should see at 20 feet. The fraction 20/30 simply means that the eye sees at 20 feet what it should see at 30 feet. The fraction 20/40 means that the eye sees at 20 feet what it should see at 40 feet. These fractions mean just that much and no more.

The trouble with this vision percentage question is that we have been taking too seriously the fractions by which we represent acuity of vision, and using them as they were never intended. Arithmetically the fraction 20/40 is half of the fraction 20/20, but the *vision* represented by the fraction 20/40 is *not* half of the *vision* represented by 20/20.

To state that a man with 20/30 vision has lost $\frac{1}{3}$ or 33 $\frac{1}{3}$ % of his vision or one with 20/40 vision has lost $\frac{1}{2}$ or 50% of his vision is absurd. In the same ratio of reduction *proportionately* 20/50 would be $\frac{2}{3}$ loss or 66 $\frac{2}{3}$ % loss and 20/60 would be total loss or blindness; which is manifestly away out of all reason. In other words if loss of 20 feet in *distance* at which one can see the object is 50% loss, or one-half, then another 20 feet of distance loss should be the other half certainly.

Percentage means a certain portion of the whole and every like portion of the whole must mean the same amount in percentage, and if taken from the whole must reduce the whole by the same definite amount, no matter from what portion of the whole it is taken.

Fifty per cent. should certainly be half way between 100% and 0%. Vision represented by 20/40 is no nearer fifty per cent. vision than vision represented by 20/60 is 0 vision or blindness.

The vision represented by and recorded as: 40/40 or 50/50 or 70/70 or 100/100 or 200/200 or 220/220 is, and are, each and every one as certainly normal vision or 100% as is that represented by 20/20. Therefore if we accept 220/220 as normal or equal to 100% and 20/220 as total loss we *must* accept the percentage as I have worked it out, viz., 1% for every two feet which we must approach the 220 foot letter in order to see it: Starting at 220 feet from a letter which should be seen by normal eye at 220 feet.

WHIP-CRACKER INJURY OF THE EYE

WITH A REPORT OF THREE CASES

EDWARD B. HECKEL, A.M., M.D., F.A.C.S.

PITTSBURGH

It has been a task to attempt adequately to express in the title of this communication its real meaning; whip-cracker injury does not express it, for the end of the whip or cracker did not come in contact with the eye. The real trauma was inflicted by a flying bit of fine brass wire which was plaited or braided into the end of the whip or what is commonly called the cracker. At first thought it is almost inconceivable how a bit of fine wire, No. 31 gauge, could penetrate into the eyeball or actually traverse the entire length of an eyeball and lodge in the posterior sclera, but nevertheless this has been demonstrated by the cases to be reported.

The whip generally used by teamsters has a long lash so that in swinging the whip it is not unlike the long arm of a lever, the velocity which the end or cracker acquires and the distance which it travels is greatly augmented and limited only by the length of the lash and the force with which it is hurled. After giving the subject of velocity some thought and endeavoring to calculate the velocity of the end of a whip, after a vigorous crack, the writer feels that he is not exaggerating in the least when he states that the ultimate velocity of the cracker is certainly more than 1,000 feet per second and perhaps nearer 10,000 feet per second. Some idea of this apparently immense velocity may be visualized when we stop to think that sound travels at the rate of about 1100 feet per second.

It has been the good fortune of the writer to see three cases the result of a flying bit of brass wire from the end or cracker of a whip. The first case was simple but paved the way for the recognition of the next two cases.

REPORT OF CASES

CASE 1.—July 13, 1910, K. K., aged 27, consulted the writer about his right eye. He stated that while driving his horse and using his whip on the previous day a bit of something from the end of his whip flew into his right eye. Examination showed that the right eye had a bit of something protruding from the cornea at the limbus at the vertical meridian above, but still

within the clear area of the cornea. A closer inspection showed the other end, which apparently was a piece of wire, in the anterior chamber. The foreign body was removed and proved to be a piece of brass wire, No. 31 gauge, 7 mm. long. The eye made an uneventful recovery.

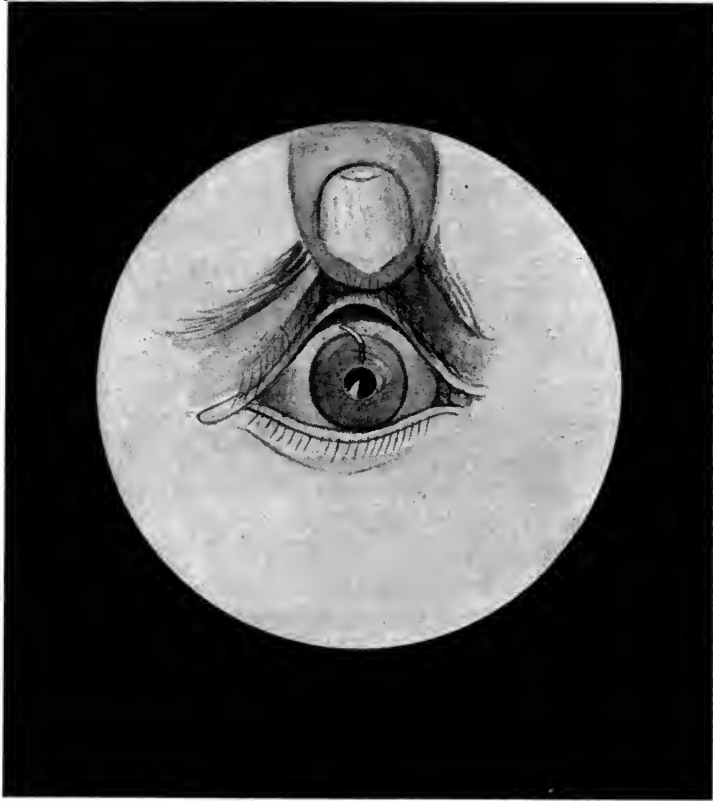


Fig. 1. Drawing showing wire in eyeball of case one.

CASE 2.—Dec. 4, 1915, M. R., aged 23, consulted the writer about his left eye which, he stated, had been injured by a piece of brass wire from the end of a whip on Nov. 30, 1915. He had been under observation and treatment from that date by some one near his home. Examination showed that an iridectomy had been done. The lens mass was still clear, the vitreous was cloudy and no fundus reflex, and vision reduced to light perception. Having in mind the experience of Case 1 and O. W. Holmes' saying, "never to guess when it is possible to know," a roentgenogram was immediately ordered to determine the presence of a foreign body. Dr. George C. Johnston made the roentgenogram and reported positive findings of what appeared to be a piece of fine wire 5 mm. long and exactly

similar to the piece removed from the eye in Case 1. The roentgenogram showed this foreign body to be a piece of wire and to be located in the posterior part of the vitreous. In view of the condition of the eye and the impossibility of the safe removal of the foreign body, an enucleation of the eyeball was advised. The patient, however, refused, signed a release and left the hospital.

CASE 3.—L. A. D., aged 28, consulted the writer Jan. 18.

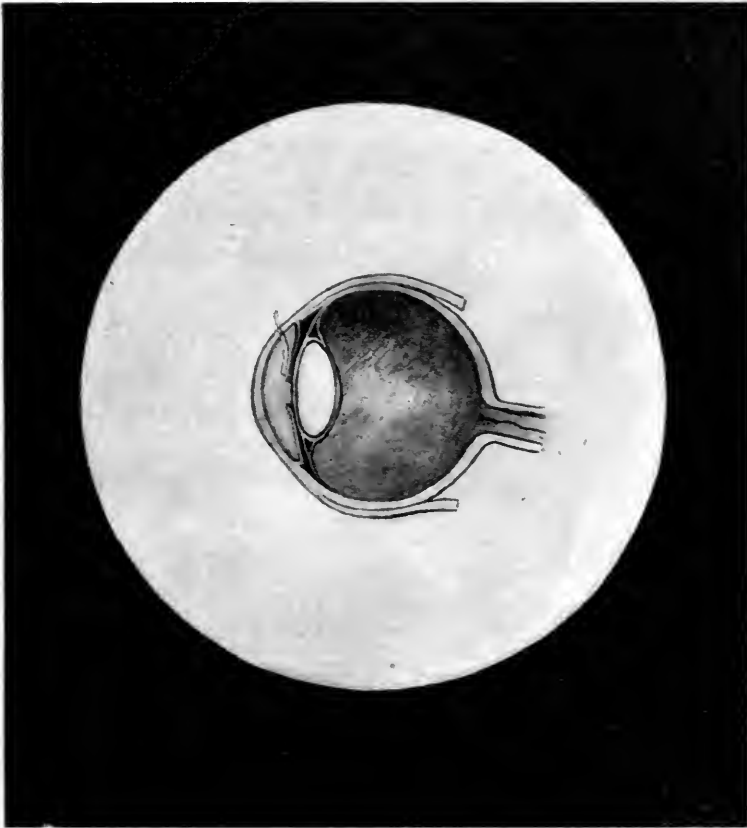


Fig. 2. Drawing showing wire in eyeball (cross section) case one.

1916, with the following history: He had had his right eye injured by a piece of something flying from the cracker of a whip in the fall of 1910. His eye was treated at that time, became quiet and remained so until July, 1914, when it again became "sore and painful." The vision by this time had been reduced to light perception. He had his eye treated at this time and it became quiet until December, 1915, when he consulted some one who suggested the removal of the cataractous lens.

which the patient, however, refused to have done. When he was seen, Jan. 18, 1916, examination showed the right eye quite pale, and no ciliary injection; cornea, clear; the iris contracted and fixed; the lens mass opaque.

Having in mind the experience of Cases 1 and 2 a roentgenogram was immediately ordered, which was made by Dr. George C. Johnston, with positive findings. The roentgenogram showed a foreign body (as per diagram) apparently a piece of wire, about

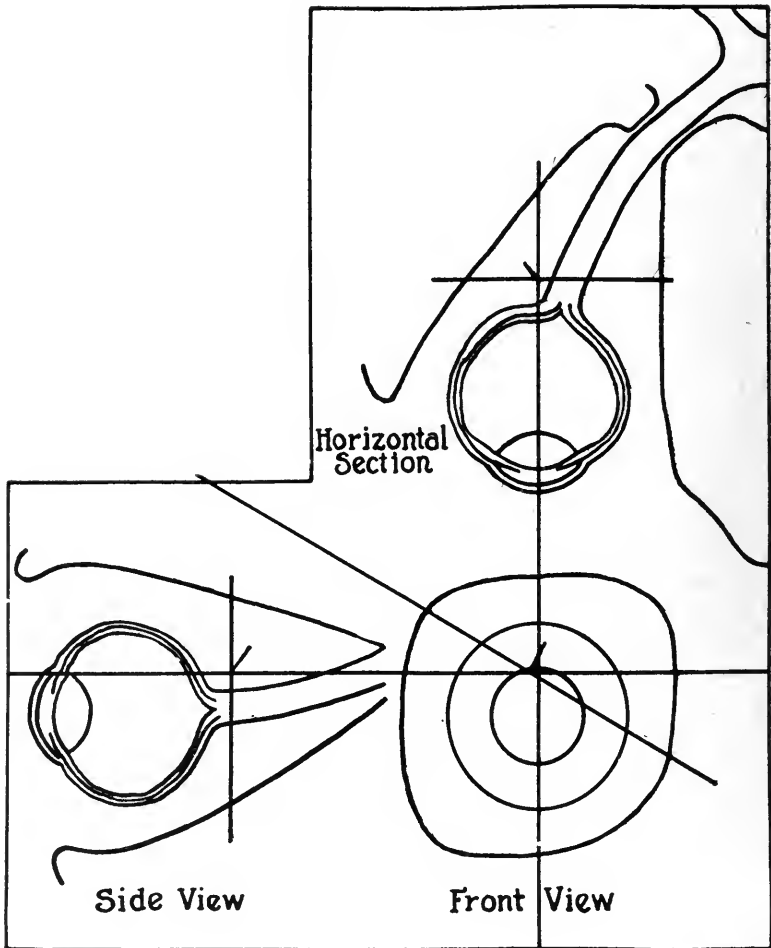


Fig. 3. Diagram showing localization of foreign body in the orbit back of eyeball in case 3.

5 mm. long and apparently the same gauge as that found in Cases 1 and 2. Localization showed this foreign body just outside of the posterior sclera. Enucleation was advised and done a few days later.

Immediately after the enucleation a search was made in the orbit for the foreign body but none was found. On examination of the enucleated eyeball, however, a dark spot was observed on the sclera at about the posterior pole, which looked very much like a bit of fine wire. This was cut into with a small knife and was suspected to be the bit of wire. The enucleated eyeball was sent to the laboratory of Dr. George C. Johnston without any history or comment whatever, but with the simple request that a roentgenogram of the enucleated eyeball be made.

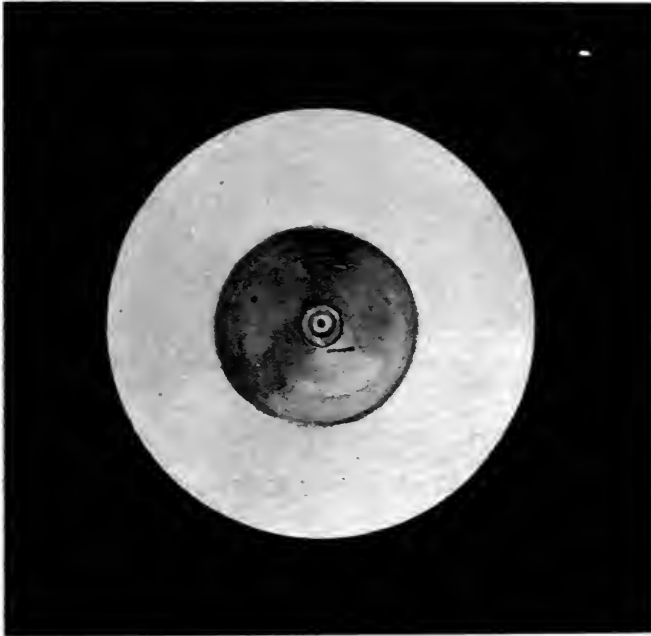


Fig. 4. Drawing of the posterior view of enucleated eyeball showing the wire imbedded in the sclera, near the entrance of the optic nerve, case No. 3.

These roentgenograms showed a bit of fine, No. 31 gauge, wire located in the posterior sclera at the suspected point. Thus demonstrating conclusively the premises and proving the immense penetrating power of so fine a bit of wire for so great a distance.

The writer has had several of these what might be called border line cases, in which a foreign body was localized at a point where it was impossible to tell accurately whether the foreign body was within or without the limits of the eyeball, so that he feels that the diagrams in use show the eyeball to be perhaps one or two mm. too small in diameter. This has been impressed so strongly on the mind of the writer that he anticipates making this a study for a separate and future communication.

The writer has made some inquiry from dealers in whips and he has learned that it is not a common practice to incorporate brass wire in the cracker of a whip, but that some whips are

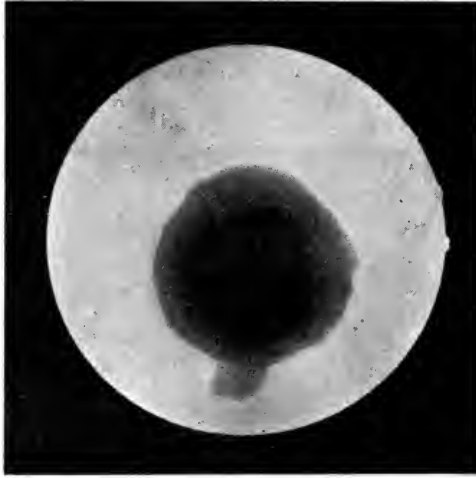


Fig. 5. Roentgenogram of enucleated eyeball, showing the wire near optic nerve. Case No. 3. (The enucleated eyeball and X-Ray plate were shown at the meeting.)

made this way, there can be no doubt as illustrated by the three cases recited. It is scarcely probable that these individuals supplied the brass wire themselves, as Case 1 was a resident of Pittsburgh, and Case 2 was a resident of Clarion County, Pa., and Case 3 was a resident of northern Allegheny County.

DISCUSSION

MAJOR JOHNSTON, Pittsburgh: The interesting thing here is that the depth to which it may penetrate in the eye has nothing to do with the size of the body. It will be determined by the mass of the foreign body times the velocity. Not how big it is, but how fast it is traveling determines how deep it will go. Yesterday the statement was made that small foreign bodies were not always recognized in the roentgenogram. Anything you can see with the naked eye can be localized in the eye, if the patient holds absolutely still, but if he moves during the exposure you will not be able to do so. To make sure he does not move the eye we continually observe that he keeps his eye in the proper direction. If he has had military training, he will do what he is told. If not, he will not do what he is told, thereby showing his independence. We determined a long time ago that all eyes are not the same size. We made some investigations and found that they vary quite remarkably, at least a mm. or two, when a single mm. will determine whether a foreign body is within or without the vitreous. There is a way to determine whether it is within or without the globe of the eye. Have the patient look in a fixed direction in a normal position of the eye axis. Then have him look well up or down and make two pictures, and have him look to the

right and make a picture. If the foreign body is in the vitreous it will move, but if within the orbit, Dr. Heckel says it will not move so much.

DR. LINN EMERSON, New York City: I will report a case illustrating what has just been said. Three years ago, just before leaving for the West, a man came in with a piece of steel in his eye. I immediately explained to him the danger and that he should have a roentgenogram taken and the steel removed; otherwise the eye would have to be removed. I took him to the hospital, and the next day left for a two months' absence. On my return I was surprised to have him tell me that the roentgenogram showed the steel in his eye, that they did not get it out and did not take his eye out. On going to the hospital I hunted up his history and his roentgenogram and found this was what had happened; (illustrating at board). The piece of steel was located as having gone through his lens and being imbedded in the optic nerve just outside the sclera. Effort was made with the giant magnet to withdraw this but they did not succeed. It was thought by several that in all probability this was embedded in the nerve outside the globe and he was advised to let it alone. The lens became cataractous and I let him go two years, and at the end of that time the eye became soft and he began to have vague symptoms of sympathetic irritation in the other eye and I explained to him the danger to the other eye and advised enucleation, which was done. The piece of steel was found embedded in the globe, just inside the sclera. So that where the foreign body apparently is just outside the sclera, there is a possibility it is not outside.

DR. EDWARD JACKSON, Denver: I want to report one case similar to Dr. Heckel's I saw several years ago. The piece of wire was imbedded in the periphery of the anterior chamber extending back through the iris. It was extracted about a week after the accident. I was informed that brass wire was frequently used in the ends of lashes of certain whips, that it stood the wear better than leather or other material. We can see that it is an enormous force that will throw off anything like that.

With reference to the diagram of the location of the foreign body in the eyeball or just outside. The diagrams are sometimes made just the usual diameter of the eyeball. The shadow cast by the divergent ray must be larger.

DR. E. J. BISSELL, Rochester, N. Y.: This point of determining whether a foreign body is just in or just out of the eye is important. In a case of mine, the man who made the roentgenogram outlined it carefully and said it was very close to the sclera but did not want to say positively on which side. With the ophthalmoscope I could see to the temporal side of the disc a little mass of hemorrhage, and it was impossible to tell whether it was in that mass or posterior. I had my assistant take a hand magnet and begin at a distance of 4 feet and keep coming closer, and pretty soon the little mass began to bulge forward. I had a roentgenogram made again and concluded it was just posterior, and as there was no irritation I decided to wait until the hemorrhage cleared up. There was no foreign body there, but with the ophthalmoscope I could see the sclera draw forward at that point when the magnet approached the eye.

DR. HECKEL (closing discussion): There are many conceivable ways in which a foreign body may be lying on the eyeball and yet be outside. We will have to discover some technic to enable us to determine it positively. I believe it is bad practice to use the magnet as a diagnostic instrument. It should never be done. In Dr. Bissell's case it did no harm, but nevertheless the magnet as a diagnostic instrument is wrong and should never be attempted.

THE SURGICAL TREATMENT OF CORNEAL SUP- URATION IN EXOPHTHALMIC GOITER

ARNOLD KNAPP, M.D.

NEW YORK

Slight corneal involvement in exophthalmic goiter is easily controlled by care and simple remedies. The severer corneal lesions, particularly when they occur in the toxic cases, are desperate and tax the surgeon's resources to the utmost. The loss of both corneae in exophthalmic goiter is not so very unusual; Sattler, in 1907, collected the reports of forty cases in which both eyes were lost. The following case of exophthalmic goiter in which one eye was lost and the other eye was saved with some vision may be of interest.

REPORT OF CASE

B. K., aged 35, stated that his eyes began to become prominent in August, 1916. His general condition during the past six months has been very much affected, and he has lost 40 pounds in weight. Jan. 13, 1917, the patient came to the Herman Knapp Memorial Eye Hospital on account of pain in the left eye. Both eyes were very prominent; the right eye only could be closed with effort, and at night this eye was kept open; there was some conjunctival congestion. The conjunctiva of the left eye was red and chemosed, the cornea was infiltrated with pus and was dried out, a little clear cornea remained in the upper part. Vision: R = 20/50; L = 1/200.

The patient was restless, anxious, with occasional tremor and a dry skin. Pulse about 90. The thyroid gland was hard, fibrous, but not enlarged. No difficulty on swallowing. He had a heart lesion and weighed 115 pounds. Dr. John Rogers of New York was kind enough to see the patient and prescribed adrenalin and advised the ligation of the left inferior thyroid artery. This operation was done. During the next few days his general condition remained about the same; at times he was alert and quiet, at other times he was restless, apathetic and could not sleep at night, and had no appetite. He had no diarrhea. The congestion of the right eye was relieved and the cornea remained intact. The process in the left eye continued and the cornea sloughed. January 20: Blood pressure 104; weight 110½ pounds. As the patient was losing weight steadily, Dr. Rogers advised transferring him to the St. Francis Hospital, where he was better prepared to ligate the remaining thyroid arteries, which was then done.

February 5, the right eye had again become inflamed and a

part of the outer wall of the orbit was resected by Dr. Rogers to relieve the intraorbital pressure. I saw the patient on February 11, as the right eye was not doing well. I found the right conjunctiva chemosed and swollen and the cornea in its lower half infiltrated with pus. The patient was immediately returned to the eye hospital and an attempt was made to arrest the process in the right cornea by frequent irrigation, applications of argyrol salve, and bandaging. February 12, the corneal condition was worse; the part of the cornea above, which had remained clear had now become clouded. There was very little discharge. The general condition of the patient was about the same, except that he had lost 10 additional pounds.

Under ether it was decided to suture the eyelids, following out the suggestion of Priestly Smith mentioned in the discussion of Juler's article.¹ An intermarginal incision was made in both lids, splitting the lids rather deeply. Mattress sutures were then introduced from above through the cut part of the lids, drawing together the bottoms of the two grooves, and the lids were approximated without much tension throughout a broad contact of the cut surfaces. Cutaneous incisions were made along the margins of the orbit above and below and on the temporal side in order to relieve the tension of the skin, as suggested by Bishop Harman². The left eye was eviscerated. At the following daily dressings the lids of the right eye looked normal, though somewhat bulging, and the sutures were apparently holding. The conjunctival sac was irrigated daily from the sides. The patient's general condition was about the same. A small opening formed at the center of the lids through which the progress of the corneal lesion could be watched. The upper part of the cornea cleared, a small perforation took place below, and a necrotic plug was cast off. February 20 his weight was 91½ pounds. On the fifth day the sutures were removed on account of stitch-infection, the lids however remained united. The patient was put on thyroid residue in addition to the suprarenal extract, and his general condition began to improve. The upper third of the cornea remained clear. Several decayed teeth were extracted, and the general improvement continued. The points of union of the lids slowly stretched, forming bands from 2 to 3 mm. long, the cornea nevertheless was sufficiently protected. The general health showed steady improvement, but there was no recession of the exophthalmus. The bands uniting the lids were divided and this fall an optical iridectomy was performed upward; the outer third of the palpebral fissure was sutured to facilitate closing of the lids. The vision was 6/200.

This desperate case is instructive from a number of viewpoints. It shows, first, that there is no general treatment which will lessen the exophthalmus promptly enough to safeguard the affected cornea. C. H. Mayo and others recommend the resection

1. Juler, F. A.: *Acute Purulent Keratitis in Exophthalmic Goitre*, Tr. Ophth. Soc., 1913, page 71.

2. Harman, Bishop: Tr. Ophth. Soc., 1913, 74.

of the superior sympathetic ganglion under these conditions. This procedure probably offers the most hope though the operation is not simple, and our patient's poor general condition did not warrant so severe a surgical intervention. Juler¹ has investigated the results of sympathectomy, and states that Balacescu³ found that after operation out of 41 cases, the exophthalmus was improved in 34 after a few hours or days, in 3 cases one eye only was improved, in 4 there was no effect. Later results were given in 25 cases, and marked improvement and even disappearance of the symptoms were observed in 19, no change in 3, increase in 3. In Juler's own case no marked recession of the exophthalmus occurred after this operation. In other words, favorable influence on the exophthalmus is by no means obtained in all cases. At the same time, in view of the desperate nature of the condition, if the patient's condition permits, this operation should be attempted.

The local condition remains the important one to correct, and if this can be relieved the corneal process will be arrested. The important contributing cause is the chemosed and thickened conjunctiva in the lower half of the eyeball, which mechanically prevents the closure of the lids. The lids must be brought together to cover the cornea. Suture of the lids has undoubtedly been practiced by every one for this purpose, but ordinary suturing even with freshening of the lid-margins is not satisfactory, as the sutures do not hold. In my opinion the suture method of Priestly Smith augmented by the releasing cutaneous incisions of Bishop Harman meets these conditions best, and the favorable result obtained in this case must be ascribed to it.

DISCUSSION

DR. D. T. VAIL, Cincinnati: I never encountered this sad complication in this disease; my experience with sloughing cornea from lagophthalmus has been limited to paralysis of the facial nerve where the lids were so widely open as to afford no protection to the cornea. In a case like the essayist's, operation is necessary. Something must be done to protect the cornea. The eye stands widely open. Sometimes the lids recede behind the ball. Priestly Smith's method just described is heroic and should be a last resort if indeed it is to avail. I have learned that the operation done for keratitis e lagophthalmo in facial paralysis we may sacrifice one-third of the lid margin without deformity after paralysis disappears. We pare the free margins of the upper and lower lids next to the outer canthus for a distance of one-third the lid margins in such a way that by deep suturing with strong thread left in situ for five days we will have firm union or ankyloblepharon established. Here is where the operation usually ceases in the hands of most operators but I have introduced another step which is very necessary for successful holding

3. Balacescu: Arch. f. klin. Chirurgie, 1912.

together of the lids. I carry out the same principle in suturing the inner third of the eyelids. I also pare the free margin of the upper and lower lids at the inner canthus to the distance of one-third the lid margin. In doing so I avoid the puncta but cut out a crescentic fold of skin overlying the inner canthal ligament above and below it and then suture the raw surfaces with strong thread same as was done at the outer canthus. I wish to claim originality for this last step for I believe it has never been described. This operation, suturing one-third the lid margins on each side and leaving the middle third for inspection of the cornea should afford sure protection for any case. The paring must be deep and honest. The stitches deep and strong. The stitches at the inner third relieve the great strain.

DR. L. M. FRANCIS, Buffalo, N. Y.: Sometimes these lids can be closed without resorting to stitching. If one cements a wedge-shaped piece of gauze to both lids, extended by a loop, and then runs silk through the loops, these loops may be tied and the lids approximated, and opened for dressings. A dressing of this sort will hold two or three days and can be replaced by an intern. This procedure was reported by me eight years ago.

MAJOR GREENWOOD: A simpler method than that of Dr. Vail's is one that has been applied in the war zone. Many soldiers have injury to the facial nerve, causing lagophthalmus, or from some cause the eye is protruding and there is danger of losing the eye by ulceration from exposure. One man came in to the base hospital with a corneal ulcer in the lower part of the eye as a result of exposure from complete lagophthalmus, due to shrapnel injury of the left side of the face. I covered the ulcer with a conjunctival flap, first removing the pus from the anterior chamber, before drawing the flap up. The lids were brought together by paring the outer two-thirds without cutting the lashes, and suturing where adhesion was desired, leaving a little opening near the inner canthus for treatment and observation. In a few days the stitches were removed. Prior to the war I had two women who came to the Boston City Hospital. Both had such marked exophthalmos that the lids could not be closed, each had lost an eye from a perforating ulcer and had ulceration of the remaining eye, and in both we obtained an excellent result by covering with a conjunctival flap over the lower half of the cornea and stitching the lids together. Where you have a lagophthalmus you want to suture them so they will adhere, to be severed later when desired.

PAINFUL ACCOMMODATION

JOHN GREEN, JR., M.D.

ST. LOUIS

In the last few pages of his epoch-making book "On the Anomalies of Accommodation and Refraction of the Eye" (Sydenham Society, 1864), Donders discusses "Spasm of Accommodation," and "Painful Accommodation." He is careful to distinguish between the normal tone of the ciliary muscle and the "exalted" tone which in hypermetropia and sometimes in astigmatism, "wholly or partially conceals the abnormal condition." He is frankly sceptical about the existence of acute spasm (comparable to the spasm produced by physostigmin) and explains his scepticism on the ground that he has "never met with a clear case of it."

The relation between the physiologic tone and the pathologic exalted tone or contracture of the ciliary muscle is discussed by Lancaster and Williams in their careful study.¹ They quote from Howell's textbook on Physiology as follows: "By tone is meant a state of continuous contraction which is slight in extent under normal conditions." Contracture is defined by these authors as "a state of maintained contraction, or, looked at from the other end, as it seems to us, better point of view, a state of retarded relaxation." As they remark, "Contracture is an excessive activity and should be stopped . . . tonus is normal and physiologic and does not need to be stopped." The task of the examiner is, therefore, to determine how much of latent hypermetropia is due to tonus, and how much to contracture, and to prescribe a glass which shall do more than "relieve the excessive action."

We are all familiar with the great variety of asthenopic symptoms, including pain, occurring in hypermetropic persons as a result of this contracture of the ciliary muscle. The existence of contracture is readily proved by the use of atropin which discloses the great difference between manifest and total error. Accommodation associated with pain due to contracture does not require comment. But there is an important group of cases in which pain, often of great severity, is evoked by accommoda-

1. Lancaster and Williams: *Tr. Am. Acad. Ophth. and Otolaryngol.*, 1914, p. 170.

tion and in which there is little or no evidence of accommodative cramp. It is this accommodative anomaly, designated by Donders as "Painful Accommodation," to which I desire to draw your attention. Perhaps a synopsis of a case related by Donders may help to make the clinical picture clear.

A TYPICAL CLINICAL HISTORY

A patient residing in the East Indies experienced pain on making observations with optical instruments. His state was considered to be hyperemia of the retina. On reading and in calculating his observations the pain increased. Under atropia, hypermetropia of 1.5 was disclosed. Convex glasses, both weak and strong, failed to relieve. Derivants and leeches were tried, with equal want of success. Reading for a few minutes produced pain which compelled him to desist. After suffering 18 months he wrote to Donders who ordered atropia twice a week and convex glasses of different strengths for work at different distances. Almost at once the pain ceased. I give the rest of the history in Donder's own words:

"When after a first disappointment, the use of atropia was, a few months later, for the second time suspended, the pain on tension of the accommodation did not return, and so far as I am aware no disturbance whatever has since recurred in making observations, reading, writing or calculating. This case scarcely needs any comment. It shows that in slight degrees of hyperopia a condition may be developed by continued tension in which the least accommodation for near objects becomes very painful. Spectacles are then of no use, because with the convergence involuntary accommodation is combined, and this again excites the pain."

Later writers have sought, and with much reason, to associate this accommodative anomaly with certain states of nervous instability—notably neurasthenia and hysteria. The modern view is well expressed by De Schweinitz², who says:

"Patients of the hysterical type are afflicted with markedly painful visual sensations, notably dread of light, lachrymation, blepharospasm, neuralgic pains which appear in the neighborhood of the ocular globe and radiate to the temporal and zygomatic regions, and imperfect eye endurance. To this set of symptoms, the term hyperaesthesia of the retina has sometimes been applied. They represent the condition which Förster described as hysterical kopiopia, Donders as painful accommodation, Nagel as hyperaesthesia of the ciliary muscle, and Schenkl as hysterical

². DeSchweinitz: Posey and Spiller, *The Eye and Nervous System*. Philadelphia, J. B. Lippincott Co., 1906, p. 640.

ocular pain, but which, in general terms, may be classified under "painful visual sensation" or "hysterical asthenopia. . . .

"Any effort of vision, particularly at close work, is quickly followed by ocular pain, brow neuralgia, and diffuse headache, often with micropsia and obscuration of vision, and quickly by fatigue. . . . We have in these cases, according to Binswanger, to deal with hyperalgesia manifesting itself through light impressions, and he believes that these hyperalgesic conditions form part of a general hyperalgesia, most pronounced in certain forms of hysteroneurasthenia."

REPORT OF CASES

CASE 1.—History.—Mrs. G. D., aged 55, consulted me, July 17, 1917, with the complaint that for the past six years she has had increasing difficulty and pain in near vision. The condition has become greatly aggravated during the past six months, so that at this time it is "real torture" to try to read. Recently, too, she has had "spells of trying to look through shadows." The patient has worn bifocals for thirteen years, has had numerous changes, the last eight months ago.

She is greatly depressed over her ocular troubles, and has about made up her mind that she is doomed to irremediable pain, if she uses her eyes, and hence has very largely given up near work. Nor is the fear of impending blindness very far in the background. She tells her tale in rather a hopeless way and clearly conveys the impression that she has lost confidence in all oculists, her present advisor included. She is continually shifting her position in her chair, throwing her head from side to side, and moving her hands aimlessly. Questioned as to bodily pain, she denies it, but says she is nervous all over and cannot keep still.

Examination.—Repeated tests indicated a compound hyperopic astigmatism, but as there was much uncertainty in regard to the strength and axis of the cylinder, homatropin was ordered. The cycloplegic test yielded:

O.D. + 2.75 + .25 cx 120 V 6/5
O.S. + 2 + .75 cx 60 V 6/5

Bifocals based on a postcycloplegic test, were ordered as follows:

O.D. + 3. + .25 cx 120°: + 2 added
O.S. + 2.37 + .62 cx 60°

The muscle balance showed orthophoria at 6 m. and exophoria 3 degrees at 33 cm. Accommodation 1 D. Ophthalmoscopic examination negative, save for a little undue tortuosity of the finer vessels adjacent to the macula. Tension, R. and L. 15 mm. Hg. Fields normal.

As my final result did not differ conspicuously from the prescription given by my predecessor, I felt assured that should I pursue the inquiry no further, I should probably be classified in the patient's mind as the latest and least successful of her oph-

thalmic advisors. It seemed worth while, therefore, to gain some knowledge of her life and ailments.

Previous Personal History.—She recited a typical story of a farmer's wife—hard physical work, and early struggle to make both ends meet, the bearing of children, and the denial of personal necessities over a long period of years in order that the children might have educational advantages. At the birth of her first child she sustained a bad perineal laceration. This was not repaired and during subsequent pregnancies she suffered extremely. A secondary repair was successfully made fifteen years later. To add to her troubles, her teeth had given her "lifelong misery." All were finally extracted, save two or three incisors in the lower jaw. Menstruation ceased four years ago.

Roentgen-ray examination of the few remaining teeth showed marked pyorrhoëa and in one an apical abscess. They were extracted. The patient was referred to Dr. F. M. Barnes, Jr., for neurologic examination. His inquiry elicited the following additional facts: On going to bed she has what she calls "nervous fits" for an hour or so, she "wiggles and turns," cannot get to sleep and experiences some disagreeable and uncomfortable sensations in the occipital region. She is inclined to worry over trifles. She has one son of draft age, but is not worried over the possibility that he may be drafted. The physical and neurologic examination proved wholly negative. Dr. Barnes writes: "It is rather hard to find a definite term which exactly sums up her condition, but perhaps a neurasthenia incident to the period of the menopause expresses it about as well as can be.

Treatment.—Ovarian extract was ordered and a strong assurance was given the patient that she would get better. She was dismissed with the injunction to use her eyes for a certain period daily, stopping the moment pain manifested itself, and to endeavor to increase the period of near work each day. It was clear that the patient had taken a new grip on life; her outlook, filled with gloomy forebodings, had been replaced by optimism.

Results.—A letter of inquiry recently addressed to the patient elicited the following reply: "After having to begin over several times, I have reached the point where I can read for an hour in the morning and again in the afternoon, but not more than fifteen minutes by artificial light. I am getting along very comfortably, eyes and general health both."

CASE 2.—History.—Dr. X consulted me, Nov. 8, 1916. In order properly to understand the present state of our patient, it is necessary to sketch, briefly, his career. After graduating in medicine he set out to achieve a large measure of success as a practitioner. The will to do was there and he had the physical capacity to make his body do his every bidding. He was successful from the start. He entered actively into the work of various medical societies. He held various teaching positions in his medical school and ultimately occupied the chair of surgery. For twenty years it was one continual grind from early morning until

late at night. Often he would come in at midnight, tired out after a round of visits and spend the next two or three hours preparing himself for the next day's lecture or demonstration. And because he had the physique he could do all these things. And so, year after year, he kept driving on, with never a thought of the break that, sooner or later, must come.

In spite of his strenuous life he supported, with equanimity, a moderate hyperopia, with a low inverse astigmatism. Without experiencing any of the signs of early presbyopia, he reached the age of 47 when, rather suddenly, the accommodative strain became too great. After a morning's operating, he would pass a wretched afternoon, with blood-shot, aching eyes. The oculist consulted prescribed reading and operating glasses with bromids internally. There was no relief, rather the symptoms increased in severity. Finally a sea trip was planned and undertaken. Not for a single moment during his five weeks' vacation were the patient's eyes in the least degree troublesome. He sedulously abstained from reading, and came back early in the autumn rejuvenated and fully convinced that his eye troubles were definitely at an end. But his hopes were dashed the first morning he attempted to operate for the eyes burned and ached. That afternoon was spent on a couch in a dark room with cool cloths applied to the closed lids. Through September and October he led a miserable existence, endeavoring to get through with the accumulated operative work of the summer, and spending his afternoons and evenings in ocular misery. He was fond of social life, but found the bright lights of the drawing room intolerable. "On several occasions," he told me, "it has required all my will power to remain in a friend's house the time demanded by the rules of courtesy." Such an experience would be followed by a sleepless night.

Examination.—His appearance did not denote a man who had been through great suffering. Rather he seemed well preserved and physically energetic. His refraction proved to be a compound hyperopic astigmatism as follows:

$$\begin{array}{l} R + .87 + .5 \text{ cx } 165^\circ \text{ V } 6/4 \\ L + 1.12 + .37 \text{ cx } 180^\circ \text{ V } 6/4 \end{array}$$

Accommodation 2.75 D, either eye. Muscle balance at 6 cm. showed lateral and vertical orthophoria.

$$\begin{array}{ll} \text{Prism adduction } 17^\circ & 3^\circ \text{ exophoria at 33 cm.} \\ \text{Prism abduction } 4^\circ & \end{array}$$

Reading glasses, as follows, were prescribed:

$$\begin{array}{l} R + 2.12 + .5 \text{ cx } 165^\circ \\ L + 2.37 + .37 \text{ cx } 180^\circ \end{array}$$

A week later the patient reported "absolutely no improvement." Distance glasses were prescribed as follows:

$$\begin{array}{l} R + .75 + .5 \text{ cx } 165^\circ \\ L + 1. \quad + .37 \text{ cx } 180^\circ \end{array}$$

A rhinological examination was negative.

The patient absented himself for a month. On his return he stated that matters had been going from bad to worse; that he had faithfully used the distance and reading correction without any appreciable effect on his symptoms. In despair he had, toward the end of the month, broken away from work and gone to his farm in a distant state. There, freed from the necessity of using his eyes, he had gotten some relief. Immediately on resuming work at home the symptoms and recurred in all their severity.

Bifocals were ordered. Astringents were used for several days to combat a slight conjunctival irritation. One month later (January, 1917), it was clear that no progress was being made. The effort to use the eyes invariably produced an intense deep-seated ocular pain, lasting all the rest of the day and often well into the night. At times it became almost unbearable, suggesting morphia which, however, our patient had the good sense to refrain from using.

Treatment.—It was now clear that we were dealing with an intensely irritable ciliary muscle and the indication was for complete physiologic rest. Accordingly, having satisfied myself of the absence of glaucoma, I prescribed atropin sulphate 1/2 per cent. *ter die*. Jan. 2, 1917, three days later, the patient reappeared, overjoyed, stating that yesterday was the first comfortable day he had had since September. His relief was complete and he was entirely willing to put up with the unpleasantness of a dilated pupil and functionless accommodative apparatus. He was kept completely atropinized (1 per cent. five times daily) for three weeks. During this period he was absolutely free from ocular discomfort. His total refractive error proved out:

$$\begin{aligned} R &+ 1.37 + .25 \text{ cx } 150^\circ \text{ 6/4} \\ L &+ 1.5 + .37 \text{ cx } 30^\circ \text{ 6/4} \end{aligned}$$

(If reference is made to the precycloplegic measurement, it will be seen that the latent hyperopia is only .5 D which is accounted for by the physiologic tone of the ciliary muscle.)

Bifocals were prescribed as follows:

$$\begin{aligned} R &+ 1. + .25 \text{ cx } 150^\circ \\ L &+ 1.12 + .37 \text{ cx } 30^\circ. +2 \text{ added.} \end{aligned}$$

All use of the eyes was interdicted for two weeks more. From this time, progress was slow but steady. In view of the beneficial effect of outdoor life (as evidenced by improvement on former vacations) the patient was urged to abandon his practice and go back to his farm. Fortunately, our views coincided and early in February, 1917, the patient betook himself to his farm, where he undertook a systematic reeducation of his dilapidated ciliary muscles. In the beginning he was limited to two minutes reading three times a day. As this amount was possible without ocular discomfort at the time or pain afterward, the periods were gradually increased. Under date of June 21, he writes: "I am still improving. The last three weeks have been better than any

similar period during the past year, except when I was not using my eyes at all for close work. I am now using my eyes forty minutes twice daily without pain."

CASE 3.—History.—Mrs. L. E. H., widow, aged 62, first seen Dec. 22, 1916, complained of "terrible" suffering when attempting to use her eyes. "The eyes feel like balls of fire."

Examination.—This showed no external ocular abnormality save slight shrinkage of the conjunctiva of the lower fornix (for this I cannot account as there was no history of conjunctival affection). The fundus was normal. Refraction proved to be a compound hyperopic astigmatism as follows:

$$\begin{array}{rcl} R + 2.25 + 1.75 \text{ cx } 75^\circ & 6/5 \\ L + 2. & + 1. & \text{ cx } 105^\circ & 6/5 \end{array}$$

Accommodation 1 D. Orthophoria for distance. 3° exophoria at 33 cm. Prism adduction 24° . Prism abduction 7° .

Bifocals prescribed:

$$\begin{array}{rcl} R + 2.25 + 1.75 \text{ cx } 75^\circ \\ L + 2. & + 1. & \text{ cx } 105^\circ; + 3. \text{ added.} \end{array}$$

Treatment and Results.—Examination by Dr. L. K. Guggenheim disclosed a subacute rhinitis and markedly hypertrophied tonsils containing pus. The left ear presented all the symptoms of a chronic adhesive process with atrophic changes in the cochlea. The condition of the tonsils was so bad that, in spite of the patient's age, their removal was deemed imperative. The operation was performed under local anesthesia, and was followed by several days of extreme prostration, during which time the patient remained in the hospital. About two weeks later she appeared at the office, thoroughly shaken, trembling all over, and complaining of great pain in the eyes and head. She was seen at intervals during the winter, her complaint being monotonously the same—utter inability to use the eyes for more than a minute. Efforts to prolong their use were attended by severe burning sensations followed by intense aching pain. The pain did not terminate with the termination of reading, but persisted into the evening and indeed all night. The patient was tractable and willing. She followed instructions to the letter. I hoped, by reading tests of short duration, beginning with one or two minutes, and gradually increasing the period, to train the irritable ciliary muscle to functionate more nearly normally. My endeavors were vain. Toward the end of June she appeared stating that for several days she had had severe pain in the left upper arm and shoulder. A doctor had told her that she probably had a "tumor." Fear of amputation and the very evident severe pain, brought her to a pitiable state of nerves and tears. She was on her way to her home town and was about to place herself in the care of her cousin, a physician. Two weeks later she reappeared, her arm and shoulder wholly recovered. Her physician, Dr.

Rendleman of Cairo, Ill., informs me that her trouble was purely functional.

During July she was kept for two weeks under atropia; relief was complete, but after the effect of the drops had worn off she was still wholly unable to use her eyes on account of pain. It seemed clear enough that I might work with the patient till doom's day and yet reach no satisfactory result.

Dr. Barnes, to whom the patient was referred, elicited the following additional data: "The present trouble began about nine years ago, at first with severe supraorbital headaches, soon after associated with severe pain in the eyes in use. Her illness has been progressive, new symptoms having appeared from time to time until now they are almost innumerable—principal among them are nervousness, fainty spells, indigestion and nausea without vomiting and insomnia. The patient talks volubly, recounts her symptoms in great detail and with many extravagant expressions regarding her sufferings. She is accurately oriented in all fields, shows no hallucinations or delusions, her insight is fair, memory unimpaired; there is no evidence of deterioration."

There was no organic involvement of the nervous system. Diagnosis: "Presenile Neurosis of the Neurasthenic Type." On account of the age of the patient prognosis "not good."

The examples of painful accommodation related by Donders were all in persons under the presbyopic age, and references to this subject by subsequent writers that I have consulted make no allusion to its occurrence in middle aged and elderly persons. I am inclined to believe that the condition is not so rare as the paucity of references would seem to indicate. No doubt many cases escape us through failure to put ourselves *en rapport* with the whole physical and psychic history of our patients. I am convinced that there is always a background of physical or mental stress (often both), eventuating, finally, in some form of nervous instability. The painful irritability of the ciliary muscle may be the sole expression of the neurasthenic state, as in the case of Dr. X., or it may be one symptom added to a multitude of other signs of nerve weakness, as in the cases of the two women. The cases related illustrate, also, differences in prognosis. Where the background is neurasthenia due to a passing physical state (as in Mrs. G. D.) or where the ciliary muscle is solely implicated (Dr. X.), the prognosis is good, although the course may be protracted. But with Mrs. L. H., the situation is different. In her the development of the neurasthenic state has been very gradual—she has passed from middle to old age, acquiring a long list of nervous manifestations. "You cannot teach an old dog new tricks," and you cannot reeducate an elderly neurasthenic. If the prognosis, from the general neurologic stand-

point, is dubious, it is not likely that the disordered ocular function can be restored to normal.

DISCUSSION

MAJOR W. B. LANCASTER: I think, as Dr. Green has said, that these cases are not very rare. The type is familiar to me though I had not thought of calling it by the name that Dr. Green suggests, or in fact by any name. I do not expect to find these symptoms unless the patient is either a hypermetrope wearing too weak convex lenses or, more rarely, a myope wearing too strong concave lenses, and the typical cases always come on after presbyopia has set in. It may be before they are 40 years old because presbyopia may show itself long before 45 in a patient who is handicapped as above, namely: a hyperope who is not wearing glasses or with too weak convex or too strong concave lenses. In other words, the accommodation has for many years been overworked and increasingly overworked, that is, he is carrying a load which he is every year less able to carry. At first such a patient is able to perform this extra accommodation with ease, it is well within his powers, for he has a wide margin of safety or reserve of accommodation. As the range of accommodation decreases, associated often with overwork or reduced strength from other causes, the margin of safety disappears. Nevertheless, eyes which have always been able to take care of this defect refuse to give in. It is in the beginning of presbyopia that the most desperate efforts are made to overcome hypermetropia which is uncorrected and it is in these persons that latent hypermetropia is most difficult to make manifest; in other words, they will not accept the lenses which would correct their defect even if they are prescribed so that the greatest skill and patience and persuasiveness are needed to bring about a cure.

It is surprising how often the break-down in these cases is comparatively sudden instead of gradual as might be expected since it is due to a gradually increasing burden, the increase of which could not be more gradual. It is simply a case of passing a critical point. Progress toward that critical point may be excessively slow and gradual but the change when that point is passed is sudden and complete. Compare water slowly cooling with no marked change till the freezing point is reached or the gradual weakening of an artery and increase of blood pressure which precede an apoplexy.

Instead of recovering with comparative rapidity after you give them suitable glasses, the convalescence is long, as it was in the case cited by Dr. Green. I recall a similar case. The patient like Dr. Green's was a doctor, an intimate friend of mine. I knew he had hypermetropic eyes but did not insist on his wearing glasses because he could see perfectly well without them and had no headaches. He had a little conjunctivitis but disliked to bother with glasses so I never insisted. Finally when the critical point was passed and he was forced to wear them they did not give immediate relief and even after several years he could not use his eyes freely with comfort.

If we were guided entirely by cases of this type we should put glasses on nearly all our patients. Fortunately this type is the exception. There is another side to this problem but just where to draw the line requires the nicest discrimination and the maturest judgment.

DR. J. R. NEWCOMB, Indianapolis: It has been my misfortune to have one or two of these patients, and as I look back I can say I never have seen a case in which there was not the factor of improperly fitted correction in previous years. My observation has been an incorrect correction for astigmatism. You have a hypertrophy of the ciliary muscles.

If you give these patients relief by a full correction, when there is a hypertrophy of the ciliary muscle and an atrophy in the muscle, etc., the result is that you have the hypertrophied muscle fully relieved, and with the effect produced in the ciliary muscle you have a muscle fatigue poisoning. The rapidity with which the fatigue is carried away determines whether there is pain. Sometimes there is only a sense of discomfort. The moment you bring it up to normal there is a marked psychic discomfort. They have a psychic equivalent of pain due to their association of former experience in close work.

DR. GREEN (closing discussion): I agree with Dr. Lancaster that these cases are not very rare, but I am convinced that they are not generally recognized. Perhaps the title may have been inaptly chosen. I should welcome a suggestion for a better one.

Donder's cases, described under this title, concerned young persons, and from his description, are not to be classed as neurasthenics. Yet the clinical course closely resembled that of my elderly patients.

Dr. Newcomb suggests that an earlier incorrect prescription may have an important bearing on the development of this condition. This may be true for some cases, but certainly not for all. My physician patient had never worn distance glasses and his first presbyopic glasses were secured at 47.

I entirely agree with Dr. Newcomb about the psychic rebellion of many patients who, in their later years, are given distance glasses for the first time. I have struggled with old ladies of both sexes, who had clean cut H + Ah, and not always have I come off victorious; but tact and firm insistence will often win the day.

In the three cases related there was hearty cooperation; in fact, the distance correction was declared to be very satisfactory. Trouble came only in attempts at close work.

FILARIA LOA

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There are various synonyms for this parasite, as *Loa loa*, *Filaria oculi*, *F. conjunctivae*, *F. diurna*, and *Dracunculus oculi*.

This parasite is not the parasite known as the *Filaria palpebralis* (Wilson, 1884) which is a nematode worm commonly affecting the eyes of horses, also found in the eyes of cattle and dogs. A considerable number of this species has been recorded in recent years in mammals and birds. The former are now grouped by Railliet under the generic name *Thelasia*, the latter or those found in birds under the general name *Oxyuris*. The *F. palpebralis* has a relatively wide distribution while the *F. loa* is endemic on the West Coast of Africa, from Sierra Leone to Benguela, but it has also been known to penetrate at least 600 miles into the interior of Africa. It was carried from Africa by the negroes to South America and the West Indies, but after their importation ceased, the disease was no longer found there.

Manson says that, at the present time, cases are occasionally met with in Europe and America, in both negroes and white men, but only in persons who have frequented those parts of Africa inhabited by the parasite. Scheube describes this nematode as follows:

"The *Filaria loa* is a threadlike white or yellowish worm of the thickness of a fine violin string. The female is from 30 to 40 mm. long, and rarely attains the length of 70 mm. The male is shorter, from 16 to 22 mm. in length, or somewhat longer. The anterior extremity is rounded and blunt, the posterior end is pointed, the mouth has no armature, but is somewhat protuberant, and the intestine straight. The caudal end of the male is bent, and possesses five large papillae on either side of the anus, and is provided with two somewhat short and uneven spiculi. The uterus of the female is filled with ova 35 microns in length and 25 microns in breadth which quite early contain embryos."

The size of the worms varies greatly in infected individuals. One case will have very large worms while another will have worms of smaller size.

Castellani and Chalmers say:

"In 1891, Manson found a *Microfilaria* in the blood of sev-

eral negroes from the Congo which differed from those already described, and which he named *Filaria* (*Microfilaria*) *diurna*, and further suggested that it might be the larva of *F. loa*. Since then this hypothesis has been proved to be correct by the observations of Penel, Prout, Henly, Brumpt, Wurtz and Kerr.. *F. loa* and *Microfilaria diurna* are, therefore, simply different stages in the life-history of the same parasite."

LIFE-HISTORY OF THE PARASITE

It is fairly well established that an intermediary host is essential to the development of the *F. loa* and that this host has a restricted geographic range. The missionaries believe the insect responsible for the spreading of the infection, is a beelike blood-sucker somewhat resembling our sweat bee and called by the natives the *Osun*. The *Osun* is a diurnal blood-sucker, draws large quantities of blood, does not hurt when feeding and bites nearly altogether around the ankles. Some are much poisoned by the sting or bite while others experience no after effects of the sting. Missionaries are advised to wear high-topped shoes and thick stockings to protect the ankles.

While some of the earlier writers say that the life-history of this parasite is entirely unknown, others assert that little is positively known about it, but refer, however, to Manson's belief that the mango-fly is the intermediary host, while other observers suspect still other intermediaries. Sir Patrick Manson, writing of the life-history of *F. loa* (refer to his recent book on "Tropical Diseases"), says:

"Of the life history of *F. loa* little is positively known. As the larval form comes into the peripheral circulation of the human host, it is very probable that, like *Mf. bancrofti*, it is liberated by some blood-sucking insect, and, seeing that it approaches the surface of the body only during the daytime, this must be a blood-sucker of diurnal habit.

"Several years ago I called attention to the so-called mangrove flies as possible intermediaries, specifying as the most probable *Chrysops dimidiata*. This conjecture Leiper has ascertained to have been well founded. During a recent visit to West Africa, with the object of ascertaining the intermediary host of *F. loa*, he examined many blood-sucking insects, including mosquitoes and a variety of other insects. He found that in *C. dimidiata* and in *C. silacea* rapid and uniform developmental changes of *Mf. loa* were effected, similar to those of *Mf. bancrofti* in the mosquito, and that they took place in the salivary glands of the insect. As to the way in which *F. loa* is acquired we must await further information. It would appear that, after it has entered the human body, development is very slow, and that probably full maturity is not attained until after several years. In many

cases the parasite did not show itself until three, four and four and one-half years after the patient had left the endemic area. In one case the parasite was extracted from the eye thirteen years after the patient had left Africa, in another the worm or worms appeared at irregular intervals during fifteen years. Manifestly it is long lived. An interesting and suggestive evidence of slow development is that, while the immature active worm is often seen in children, the larval form in the blood is found as a rule only in adults. Annett, Dutton and Elliot, in 390 active children of all ages up to about 18 years, examined in a district where *F. loa* was exceedingly common in adults, found *Mf. loa* once only in a boy (aged 11). Meinhof reports a case in which the period elapsing between infection with *F. loa* and the appearance of embryos in the blood was at least seven years."

Vail reported, in 1903, that he had removed an adult worm from lower lid two and one-half years after patient had left Africa. Ferrium and Prelat removed adult worm five years after a patient had left the endemic area. All the cases, seven in number, that I have been able to see and get definite information concerning, were missionaries or members of the missionaries' families located at Kamerun on the West Coast of Africa, 200 miles west and south of Sierra Leone.

Two of the cases went to Africa in 1895, a minister and his wife. The latter became infected and had the first adult worm removed in 1899. The microfilaria were not found until 1904 or eleven years after she had gone to Africa. This patient never had any calabar swellings but has had numbers of worms removed from the eyelids and chest. She still feels the worms and her blood is swarming with microfilaria. The husband had numerous swellings but did not have adult worms removed until 1903 or eight years after going to Africa. The swellings had bothered him for several years before he felt the worms.

Their two children born in Africa have both had calabar swellings and occasionally felt the worms but no larval forms have been found in the blood. A sister-in-law of the patients just mentioned has been greatly annoyed by the worms and some very large worms have been removed, there are numerous swellings on all parts of the body but no microfilaria in blood. The other four cases had but few swellings and no microfilaria.

PATHOGENESIS

Regarding the pathogenesis, Manson writes as follows:

"As already stated, *F. loa* during the period of its growth and development in man makes frequent excursions through the subdermal connective tissues. It has been noticed very frequently

beneath the skin of the fingers, and it has been excised from under the skin of the back, from above the sternum, from the left breast, the lingual frenum, the loose skin of the penis, the eyelids, the conjunctiva and the anterior chamber of the eye. Ziemann says that it may wander about the scalp. The parts most frequently mentioned are the eyes, and although the worm may have attracted more attention when in this situation, it seems as though it has a decided predilection for the eye and its neighborhood. A patient informed me that the average rate at which a *F. loa* travelled was about an inch in two minutes. Both he and others have told me that warmth, such as sitting before a fire, seemed to attract them to the surface of the body. As a rule, the migrations of the parasite give rise to no serious inconvenience, but they may cause prickings, itching, creeping sensations and, occasionally, transient edematous swellings, 'Calabar swellings,' in different parts of the body. When the parasite appears under the conjunctiva it may cause a considerable amount of irritation and congestion, there may be actual pain even, associated with swelling and inability to use the eye and, perhaps, tumefaction of the eyelids. Should a loa wander into the vicinity of such a situation as the rima glottidis or the urethra the consequences might be serious."

All worms that were removed from patients seen, were extracted from the eyelids or chest. The rate of movement was slow but they could very quickly get too deep into the tissues to be seen. The eyelids and chest of one patient had numerous cicatricial lines that evidently outline paths frequently traversed by the worms. Cold air causes them to go away from the surface while heat attracts them.

The "calabar swellings" are edematous areas itchy and somewhat painful, lasting for three or four days. They occur frequently on the hands but may occur in any locality. It would seem that those who have "calabar swellings" do not have microfilaria.

Scheube writes that sometimes the *F. loa* migrates from one eye beneath the skin at the root of the nose to the other eye, and that one observer (Roth) also believes that it sometimes reaches the nasal canal and is then either swallowed or spat out.

TREATMENT

Under this heading Scheube says:

"The treatment consists in the removal of the parasite, which, however, is often a very difficult matter on account of its rapid movements. Should it be situated beneath the conjunctiva, the eye should be cocainized, the globe steadied by the finger, a fold of the conjunctiva over the worm raised by forceps and incised

by scissors, when the worm can be extracted by means of an iris forceps."

There is no treatment of any value known. Everything has been tried with no effect. One patient after two intravenous injections of salvarsan thought that the movements of the worms were less bothersome for a time.

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DISCUSSION.

DR. T. D. VAIL, Cincinnati: In 1905, I read a paper on this subject before this society and reported a case. I made the claim that mine was the second case in America in which the worm in question was extracted from the eye. The worm seems to have a predilection for the loose connectival tissue about the eyes, hence they are sometimes spoken of as "eye worms." It does however migrate all over the body and is only troublesome when it passes over places where the bone is close to the skin. The negroes in Kamerun, West Africa, where the missionaries have been stationed, have been removing these worms by using a thorn and sometimes they drive back the worm by means of a grain of salt in the eye. The patient has no symptom except the feeling of the worm crawling around in different parts of the body. Sometimes there are dozens in the same individual. The patient from whom I extracted this has had over a dozen removed by her husband, who is a physician. It is now twelve years since the patient resided in Africa and her husband states she still harbors four that he knows of. It is a gruesome thing to think that we may carry around in our bodies these unwelcome parasites. The theory advanced by Manson is that a fly or mosquito sucks the blood of an infected victim and in doing so takes up a number of the embryos of these worms. These embryos are smaller than blood disks and will circulate freely through the smallest capillary. They are ovoid in shape I believe and contained within a distinct capsule. The embryo worm is twisted within this hyaline capsule something like the twist of a common pretzel. Nine or ten days within the body of the intermediate host are required to have the cycle complete for the birth of the worm. Then the fly deposits on the water the embryos ready

to hatch. The water is taken in the stomach of a warm-blooded animal like the human and as soon as they strike the warmth of the stomach walls they hatch out and go crawling through the stomach wall into the connective tissue of the body. They are male and female and propagate within the body. The female discharging myriads of embryos which as just stated will only hatch after preparation in the stomach of some suctorial insect and reintroduced in the body in the contaminated water. The one I extracted was full sized. I sent it to Professor Ward, a noted helminthologist of St. Louis; it was examined and reported on by him.

I had thought "eye worms" were certainly something entirely new but was astonished at the number of references in Ward's bibliography. One hundred and twenty-seven references furnished by Ward were published in my paper.

ESSENTIAL SHRINKING OF THE CONJUNCTIVA WITH A REPORT OF TWO CASES.

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There need be no apology for reporting two cases of essential shrinking of the conjunctiva, inasmuch as the disease is exceedingly rare. It was stated by Marple that Herman Knapp in his extensive experience saw but one case of this disease. It was our good fortune to have had the opportunity of observing two cases recently. Both were in boys, one 11 years of age, colored, and one 17 years of age, white. According to Parsons one of the earliest cases of pemphigus, which is the forerunner of essential shrinking of the conjunctiva, was described by White Cooper in 1858. Before and since that time it has been confounded with xerosis. Xerosis conjunctivae is but a symptom occurring in various conditions. Essential shrinking is one of these. Those cases of essential shrinking without a previous history of pemphigus seem to show the greatest chronicity. Those with an antecedent pemphigus may evidence a rapid shrinking and obliteration of the fornices, as in the cases of Conlon and Stieren. The dryness of the conjunctiva results from the occlusion of the lacrymal ducts. Pusey in his textbook speaks of pemphigus of the skin as a rare condition constituting one to two per mille of skin diseases. Pergens states that in Vienna in the service of Kaposi there was one pemphigus case to 300 skin cases or one to 2,800 of all kinds of cases in the Allgemeines Krankenhaus.

ETIOLOGIC FACTORS

The name pemphigus is applied to a group of conditions characterized by the spontaneous development of vesicles on the skin and mucous membrane usually in successive crops. An acute and chronic type of pemphigus are distinguished, the former rare. Nothing definite is known regarding the cause of pemphigus. It is not contagious and heredity is not a factor. It has been observed after chilling. It has followed nervous disturbances as evidenced by pemphigus neuroticus and pemphigus hystericus. Similar eruptions have been frequently noted in connection with peripheral and central lesions of the nervous

system. In acute pemphigus toxic disturbances appear to be a factor, and it seems to be clearly shown in a few cases to have been introduced by vaccination or to have come from animals. Stieren's second patient had been vaccinated four weeks previous to the development of a large bleb on the forearm. This was followed by others. The nose was also affected. A number of cases have been noted in butchers, and handlers of animal products, such as hides. Dartigalongue thinks the disease is due to an unknown nervous cause.

Bacteriologically there is nothing typically etiologic found. Bulloch demonstrated, in the fluid of unruptured bullae in Pernet's and in Hardy's case, a diplococcus larger than the gonococcus; a similar diplococcus was found by Demme and by Bleibtrau in their cases. Speciale-Cirincione found a diplococcus belonging to the same family as the diplococcus of Fraenkel.

DISTRIBUTION

Any part of the body may be affected and the eruptions may occur on a mucous membrane. It is a matter of diagnostic importance to know that the disease may appear *first* on a mucous membrane. It is in fact at times confined to the mucous membrane, especially of the mouth and nasal passages. In addition to the mouth, lesions of pemphigus occur infrequently in the pharynx, larynx, nose, eyes, vagina, and presumably the stomach. In such instances adhesions between denuded surfaces are prone to occur, and in the nose have been known to produce complete occlusion. Various authors, among them Stelwagon refer to the involvement of the mucous membrane of the eyes, mouth, nose, throat and larynx, some of these occurring simultaneously. Meneau gave an extensive review with bibliography of different forms of pemphigus involving the mucous membranes, especially of the conjunctiva, nose, mouth, throat, and larynx. The occasional involvement of the buccal mucous membrane is well illustrated in Conlon's case. His patient was able to open her mouth but half way, owing to the cicatrization of her buccal mucous membrane. Conlon mentions Trautmann as having seen a patient whose mouth was completely closed for eighteen years from the same cause.

Steffan saw one case of pemphigus of the eye among 84,000 eye cases, Cohn one in 50,000, Schöler one in 50,000, Horner three in 70,000, Bäumlér seven in 97,000, Pergens two in 22,000, Franke five in 45,000; on an average one in 20,000 eye cases.

Pergens collected 133 cases but excluded 21 either as doubtful or as repeated observations. He divided the cases into four groups: (1) Those with vesicles on the skin and finally on the mucous membrane. (2) Cases without vesicles on the skin but with them on the mucous membrane. (3) Cases in which vesicles were seen only on the eye. The connection here with pemphigus cannot be positively asserted, neither can it with certainty be excluded. (4) Those cases in which no vesicles whatever are found. These are instances of true essential shrinking. In regard to these, Pergens thinks the associating of them with pemphigus is unassured, and for some entirely wrong. There were sixteen such cases in his series.

With some temerity we offer a somewhat different classification of cases presenting essential shrinking: (1) Those cases with skin lesions or blebs on mucous membranes other than those on the eye. (2) Those associated with so-called syphilitic pemphigus or syphilitic conjunctivitis. (3) Those with no antecedent history of skin or mucous membrane involvement—essential shrinking.

SYMPTOMS

In acute pemphigus severe conjunctivitis and swelling of the eyelids have been observed. Such cases are reported by Von Klemm, Senator, Seggel, and Franke. The usual form of pemphigus is the chronic variety. The eye complications are usually put under this heading. By far the best description of pemphigus of the eye is given by Groenouw in Graefe-Saemisch. From it we have quoted liberally and to it we are much indebted.

The clinical picture is variable. The disease may be ushered in as a more or less severe conjunctivitis. Occasionally a croupous like membrane is present, after the disappearance of which, an ulcer may develop. On the other hand, the conjunctiva may remain relatively intact. Subsequent to ulceration considerable scar formation occurs. The size of the vesicles on the eye varies from that of a pea to that of a bean. They may occur on the palpebral conjunctiva, fornix, or ocular conjunctiva. Their roof consists of only one layer of epithelial cells and is soon destroyed. Vesicle formation is observed according to Uhthoff, in but about one-seventh of the cases—Morris and Roberts found them 12 times in 28, Pergens 16 times in 68 cases.

Michel states that the disease attacks by preference two places of the conjunctiva, namely, the region of the inner canthus and the lower half of the scleral conjunctiva. The further course

of the disease proceeds regularly with scarring and shrinking. The first changes seem to appear constantly in the upper eyelids.

There is a tendency to confuse the disease with trachoma. Pterygium-like formations are noted. The upper and lower lids may be joined directly to the corneal limbus. There may finally result distortion of the lids, trichiasis, entropion, and rarely ectropion, the latter the result of cicatrization and shrinking of the skin of the eyelid. Ankyloblepharon is observed especially at the inner commissure or canthus. Reich and Schöler found the same at the outer canthus. Schiller says that a slight or extensive ankyloblepharon at one or both angles is seen in every case of pemphigus and never in trachoma. Cohn noted a total ankyloblepharon. In this case the conjunctival sac was intact and a severance of the ankyloblepharon led to a restoration of normal conditions.

According to the fifty-six eye pemphigus cases collected by Becker there was little or no shrinking of the conjunctiva present in 9.3 per cent., symblepharon in 19.4 per cent., complete cicatrization of the lids in 5.35 per cent. and a marked shrinking in 25.5 per cent. Both eyes are always affected but not necessarily at the same time. Several years may elapse before the second eye is involved. Michel demonstrated a case of one-sided shrinking of the conjunctiva with vesicles on the palate and epiglottis. He concluded that the conjunctival shrinking was due to pemphigus.

The cornea becomes involved at varying times. According to Pergens in about one-seventh of all cases it remains quite intact. In far advanced cases the cornea is hazy, its surface dull and dry. Vesicles rarely form on the cornea. These were observed by others, among them Täuffert, Schöler and Eales. Ulcers may develop on the cornea without preceding vesicle formation.

The element of syphilis obtrudes itself in the discussion of the condition under consideration. Hesse in the *Wiener klinische Wochenschrift*, 1915, stated that in eleven cases of pemphigus vulgaris and dermatitis herpetiformis, eight cases gave a positive Wassermann reaction, all of which reacted strongly or medium positive. With the exception of one case all were shown to be from their history and clinically, nonsyphilitic. From these findings Hesse believed it was proper to assume that the Wassermann reaction is characteristic for pemphigus. Ernest Nathan, on the other hand, found the Wassermann reaction without

exception to be negative in twelve cases of pemphigus vulgaris and vegetans.

Cases are reported in a number of instances and designated as syphilitic pemphigus of the conjunctiva. In such instances it would be of doubtful correctness to apply the term essential shrinking, though the end-results may be clinically identical. Marenholtz thinks another form of pemphigus, namely, pemphigus neonatorum syphiliticus, can lead to conjunctival pemphigus. His observations were made on an hereditary syphilitic child with essential shrinking of the conjunctiva. In Stieren's first case the patient was a syphilitic and had been taking enormous doses of potassium iodid in addition to bichlorid of mercury. An interesting speculation in regard to Fehr's case was:

"Ought this syphilitic infection to be regarded as the direct cause of the severe pemphigus of the conjunctiva and the subsequent shrinking, or as an indirect cause only, by lowering the vitality of the tissues and undermining their resisting power?"

Shumway thinks the question of syphilis in these cases is important to bear in mind. His patient had a positive Wassermann reaction and a perforation of the septum. Golowin, in a marked case of pemphigus of the conjunctiva, found a beginning atrophy of the optic nerve. This latter may have been of syphilitic origin. One of our cases happened in a congenital syphilitic.

Fromaget and Lavie report a unique case. The symptoms were multiple anesthetics, amblyopia, dryness of the eye, epiphora, blue edema of the face, nystagmus, and crises of grand hysteria. Enormous bullae occurred on the palpebral conjunctiva and the eyelids.

PATHOLOGIC ANATOMIC FINDINGS

These vary considerably. Borysickiewicz found croupous diphtheritic membranes on the conjunctiva without preceding vesicle formation. Stattler noted an enormous swelling up of the connective tissue fibers and expansion and filling of the tissue spaces with fluid. Gelpke found failure of the epithelium and a massive exudate into the thickened subconjunctival tissue. Sachs-alber described a thickening of the epithelial layer of the conjunctiva. Unthoff found besides inflammatory changes in the conjunctiva, a lifting up of the superficial layers of the epithelium yet with no vesicle formation of its own.

Great stress is laid by most writers on the conjunctival shrinking following pemphigus. In reality this is not an essential shrinking but, according to Adam, a cicatrization of the sub-

epithelial and adenoid layer of the conjunctiva. From this it would appear that the term "essential shrinking" is a misnomer and that the appellation is more strictly applicable to the slow chronic process which characterizes the disease without antecedent pemphigus. Franke found among other changes a chronic inflammation of the subepithelial and adenoid tissue of the conjunctiva.

The prognosis is almost without exception exceedingly bad. Nearly every therapeutic agent from grafting to fibrolysin has been used with little or no success. Holmström observed four cases and thought that the arsenic treatment which was instituted brought the trouble to a temporary standstill. In the hands of others the treatment with arsenic has been without avail. Bane reported a cure from the use of roentgen rays lasting two years when reported. Subsequently pemphigus developed in the other eye. Okuse after trying various unsuccessful measures, obtained success from the use of an oil derived from cooking dried nuenaugen. E. and F. Landolt report the cure of a patient who had extensive pemphigus and in whom entropion and distichiasis of the lower lids had been produced. Pedicle flaps were used; a relapse occurred at the end of three years which was remedied by a pedicle flap introduced through a canthoplasty.

Both of the cases we wish to present suffered with their ocular trouble very early in life and came under our observation years after the disease had appeared.

REPORT OF CASES

CASE 1.—S. J., now a young man, aged 17, is a student in the Missouri School for the Blind in the service of Dr. J. W. Charles. His mother, a very intelligent person, gives the history of the boy having been vaccinated when 7 months old. Two months later, blisters containing pus and blood, of about the size of the little finger-nail, appeared on the arms above the elbow, chest, abdomen and back. These the mother called chickenpox but said they had lasted for two or three months, "all of the summer." The baby was breast-fed at the time. Accompanied by fever, the eye trouble started in both eyes while the eruptions were present on the body and arms. This began with a discharge in eyes, which was followed by a dry grayish appearance of the eyeball.

The right eye has vision of O. It presents an appearance of megalocornea due to a staphylomatous bulging at the limbus. Above, the pigment shows through the stretched sclera and cornea, whereas below the cornea is densely opaque. The eyeball is almost fixed in position by the cicatricial bands, being able to move up and laterally incompletely, but not downward at all.

There is almost complete obliteration of the upper and lower fornices. The eyelids in the outer fourth are joined together by cicatricial membranes. On looking upward the eyeball carries the lower eyelid with it. Eyeball in position of divergence. Tension by finger test is markedly plus in this eye.

The vision of the left eye is 3/96. The cornea in its lower third is opaque and there is also a small central opacity. The lower fornix is almost obliterated although considerable upper fornix exists. Good rotation downward is possible but movements of the eyeball in either direction are limited. The fixation of the eyelids when the eyeball is rotated throws the conjunctiva into pronounced bands. No distortion of the eyelid edges occurs although the line of the lashes deviates inwards. Anterior chamber deep; pupil reacts readily to light. Tension by finger test apparently normal. The conjunctiva and corneal epithelium of both eyes have the characteristic dry lusterless wrinkled appearance of essential conjunctival shrinking. Wassermann test of the blood at the Washington University Medical School was negative.

CASE 2.—O. P., a colored boy, aged 11, first presented himself at the Washington University Eye Dispensary (Service of Dr. Hardy) in August, 1917. Vision was light perception in either eye. The condition in each eye was very similar, so that both eyes can be described together. The cul-de-sacs below were quite shallow and at each outer canthus had disappeared. Corneae are opaquely grayish-white over their entire extent, and in the lower inner quadrants both corneae showed dense whitish areas of opacity. All the conjunctiva was dry, wrinkled and extended onto the corneae for several millimeters all around. Both upper eyelids were readily everted. The conjunctiva over the tarsi is not changed to the same extent as elsewhere, and particularly that of the left which looks fairly normal. All movements of the eyes are good. The child is below par mentally. Wassermann test strongly positive. The mother states that when the boy was 1 year old an eruption like mosquito bites occurred on the scalp, legs and feet, in the fall and lasted about two months. Some hair was lost from the head as a result of this eruption. This eruption occurred annually and lasted through September and October for six years. It was usually accompanied by marked itching and exacerbated by scratching. Eye trouble began when 3 years of age in September, when the eruptions mentioned were present. This began with watering and discharge in the eyes which lasted for about one year, when a doctor prescribed some powder, the mother says, which "dried up the secretion." At 5 years of age the child could see no more than it does now. The father is said to have worked around racing stables and handled horses during the early years of the child's life.

It has been our endeavor to collect the references to all of the literature bearing on this subject subsequent to Pergens monograph in 1901, and the bibliography of Groenouw in Grafe-

Saemisch in 1903. It is quite possible that some of the references escaped us. A further and more careful analysis would probably reveal the fact that a number of the cases reported as pemphigus or essential shrinking of the conjunctiva are incorrectly recorded. Furthermore, as Pergens found, it is quite probable that some of the cases were reported by more than one observer. No attempt was made by us to ascertain this because of the limited time at our disposal. It appears, however, that the recorded cases in late years have been disproportionately large in comparison with the earlier statistics. Yet the disproportion may be more apparent than real and the true ratio still be as before, 1:20,000 of all eye cases.

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DISCUSSION.

DR. ARNOLD KNAPP, New York: Stated that the two cases which Dr. Hardy presented and the one which Dr. Stieren demonstrated all belong to what is spoken of as acute pemphigus. He has never seen cases of this kind. In the eye clinics of our larger cities not infrequently cases of chronic pemphigus are seen in which there are evidences of ulceration in the conjunctiva, and in some cases ulcerations in the mucous membranes of the upper respiratory tract. Another variety, and one which is the most interesting because it is so baffling, is the one to which the term "essential shrinkage," in his opinion, should be applied. In this there are no signs of either active or past ulceration to be seen either

in the eye or in any other part of the body. He is not able to throw any light on these very remarkable conditions; he has never felt like urging an operation, but it has seemed to him in the mild cases the patient was made more comfortable by the use of oil drops (olive oil or peanut oil).

DR. E. E. BLAAUW, Buffalo: I would like to speak of the boy presented here this afternoon. If that is pemphigus I will gladly accept the diagnosis. But it is not essential shrinkage. That is different. It is a disease which is very rare. I have had the fortune to have under my observation one case, a lady, aged 65, but the men of longer experience than I did not know what to make of the diagnosis. (Patient was presented at the Buffalo Ophthalmological Club last winter.) Here is a shrinking of the conjunctival sac, which becomes narrower, the conjunctiva becoming thinner and forming strands which connect the periphery of the conjunctival sac with the limbus. The remaining conjunctiva is rather loose and easy to form folds. Unless some complication, for instance, trichiasis, the process seems to stop at the limbus.

The blood of the patient was examined and found normal, no eosinophilia.

DR. EDWARD STIEREN, Pittsburgh: I will refer but briefly to my two cases quoted by Dr. Hardy; a full report will be found in the Transactions of the American Ophthalmological Society for 1915. The first one was a syphilitic and his eyes became affected during an attack of bullous dermatitis of his entire body.

The odor from this condition was so overpowering that incense had to be burned in the room when he was being dressed. There was some improvement under mercury internally and dionin.

My second case, shown by Dr. Krebs today, was followed four weeks after vaccination by a general bullous eruption of the skin affecting also the conjunctiva. Bowen, who reported, in 1901, six cases of bullous dermatitis following vaccination in children, states: "I have emphasized the fact that I cannot prove that they were caused or influenced by the vaccination."

In my opinion treatment is hopeless unless there are positive syphilitic findings, in which case antisyphilitic remedies may be of benefit.

DR. S. D. RISLEY, Philadelphia: He thought the occurrence of pemphigus in the eyes must be extremely rare, as he had seen but two cases. The first was in an old feeble man, a rag picker; he was blind barring a mere perception of light, the cornea and conjunctiva in both eyes presented much the same appearance as seen in xerosis, and there was nearly complete ankyloblepharon. Indeed, I first made the diagnosis of xerosis, but the peculiar and offensive odor led to an inspection of his skin and the discovery of an extensive distribution of pemphigus eruption. No treatment was of any avail.

The second case was a lady in comfortable circumstances seen in consultation with Dr. Randolph of Baltimore, who had been treating her for a rebellious condition of her conjunctiva and cornea. There was a slight superficial vascularity of the corneal surface throughout its lower half and slight tense folds in the conjunctiva of the lower half of the ball, extending from the corneal margin to the retrotarsal fold which were dragged on with every movement of the eyes. The condition was peculiar and quite puzzled me for diagnosis until she quite accidentally turned the eyes downward and, the upper lids following the movement of the balls unfolding the orbital sulcus, disclosed a single pemphigus sore in the skin of the lid, which I thought fixed the diagnosis of pemphigus in the early stages as an explanation for the above

described conditions. When last heard from the condition had proved rebellious to treatment.

DR. HARDY (closing discussion): The most interesting feature in this connection is the possible relation of the condition to vaccination, and it is always well to bear in mind this procedure as a probable etiologic factor. The occurrence of so-called syphilitic pemphigus is worthy of note; whether this is a true pemphigus or is in any way associated with essential conjunctival shrinking I am not prepared to say, though it would seem from some of the published reports that syphilis is an indeterminate factor.

A STUDY OF THE EYE IN DEMENTIA PRAECOX

FREDERICK F. TEAL, M.D.

LINCOLN, NEB.

In presenting this subject for your consideration, I am mindful of the fact that the majority of oculists see comparatively few cases of dementia praecox, and it might readily be concluded that a discussion of the eye in relation to dementia praecox is, to all intents and purposes, academic. But, if we believe all we are told by later day investigators, this disease is and must continue to be on the increase particularly if it is to be explained on a syphilitic basis. If, therefore, oculists can point out certain definite changes in the eye as pathognomonic of the early stages of the disease, and our knowledge of the causative factors becomes equally sure, the importance of the investigation of the eye in suspected mental deterioration takes on a new aspect.

At the time I took up my investigation, I did not realize that the field had been covered as well as it has. In Europe, the subject has been taken up by Kuhnt, Blin, Mayer, Reichman, Bumke, Murchie of Glasgow, and others; while in this country, Posey and Spiller, Tyson of New York, Thomas of Oakland, Calif., and Cooke of Seattle, have made considerable investigation in this field. They largely directed their attention toward the discovery of a syndrome of the eye in dementia praecox.

Spiller and Posey, in "The Eye in Nervous Diseases," conclude that there are no ocular phenomena of importance, no pupillary changes peculiar to any mental disease, and go on to state that where actual and persistent pupillary changes are found to exist, we can assume the presence of grave or organic disease. After discussing the investigations of other workers of the various pupillary reflexes and other phenomena, they conclude:

"That in dementia praecox definite changes in the pupillary reactions are not found, save first, such as can be attributed to defective innervation, i. e., the dilatation of the pupil; and, second, such changes or deficiencies of action as depend on diminished psychic activity."

DISC CHANGES

In 1908, Tyson wrote an article on "The Ocular Disc Changes in Dementia Praecox," and another in 1912 on "The Eye Syndrome in Dementia Praecox," working in collaboration

with Clark. His data, description and conclusions are clear-cut and forcibly presented, but have not been confirmed by other observers, except in part. He lays great stress on the disc changes, basing his conclusions on a carefully studied group of 115 cases of dementia praecox. Accompanying the article are three admirable water colors illustrating the different phases of a low-grade optic neuritis or perineuritis. The first shows congestion of disc, hyperemia and edema; dilated, dark-colored veins, with contracted arteries, and blurring of the edges of the disc. The second shows congestion of the nasal side with temporal pallor of discs, while the third shows pallor of discs with dilated veins and contracted arteries. He asserts that these different pictures are found practically only in dementia praecox. He also examined the pupillary changes in eighty-five cases. Here his observations were just as positive as they were in the fundus changes. There was a constant enlargement in the size of the pupil; the sensory reflex was absent in seventy-nine instances, and the psychic reflex in eighty-five cases.

The visual color-fields were examined in eighty-one cases. All were concentrically contracted from 30 degrees down to nothing, the average field being 10.6 degrees. He admits, however, that there was cause for error in the taking of fields, due to the lack of cooperation, in numerous instances, on the part of the patient; and the results of these examinations were included in his averages, which makes his conclusions in regard to the fields somewhat unreliable. In summing up, he says:

"The changes in the discs, pupils, visual fields (and corneal sensibility), when taken together, constitute the new syndrome and are all in accord with each other. In our examination of all other types of insanity, imbecility or idiocy, we have found no other condition similar to what we have outlined here for dementia praecox. The . . . findings indicate that dementia praecox is attended by such an early and constant syndrome of alteration of disc, visual field, pupil and corneal sensibility as to materially aid in diagnosticating this psychosis."

Tyson's investigations would seem to have settled the matter of a pathognomonic syndrome of dementia praecox, but Thomas of Oakland, Calif., while confirming his investigations in part, seems to have found practically the same disc and fundus changes in a number of forms of insanity. In an article published in the *American Journal of Insanity*, in 1914, entitled, "Optic Neuritis and the Color Fields in the Diagnosis of Syphilis, Neurasthenia, Hyperthyroidism, Dementia Praecox, Maniac Depressive

Insanity and Third-generation Insanity," Thomas, as he expresses it, takes a bold plunge. He argues that our preconceived ideas of the appearance of the normal disc are wrong; that minor and lesser degrees of optic neuritis are entirely overlooked and classed as normal. After describing his conception of the appearance of a normal disc, he pictures and illustrates seven stages of disc changes, ranging from the normal to a well developed papillitis. He lays great stress on the presence or absence of the porus opticus, or physiologic cupping, saying, "I believe that every disc without a porus opticus or with a faintly marked one is abnormal, and should be so considered." The stages referred to illustrate the natural cupping, gradual filling of cup, no cup (causing a flat appearance of disc), slight elevation, distinct swelling, and finally disappearance of the sharp outline of the disc merging into the fundus.

Thomas examined a great many cases of different forms of insanity, and asserts that he found the low-grade optic neuritis in all forms of them. He speaks of Tyson's work; and while confirming to some extent his observations, he does not admit that they are peculiar to dementia praecox. He explains these changes noted almost entirely on a syphilitic basis. He gives illustrations of color-fields. They show a contraction, most of them sectorlike with interlacing color-fields, and were the result of examination of seventeen cases, including three acquired syphilis, acquired tuberculosis, ocular neurasthenia, exophthalmic goiter, maniac depressive insanity, hypophyseal disease, angioneurotic edema, and neurasthenia. The original diagnosis was made from the above eye findings, and the etiology given as syphilis.

The conclusion to be derived from the article of Thomas is that in dementia praecox, as well as in other forms of insanity we have a syphilitic infection, acquired or hereditary, producing an optic neuritis of low grade, which is frequently overlooked; that this optic neuritis, shown by one of the various stages of optic disc change, with the sectorlike contraction of the color-fields, means syphilitic infection in the great majority of instances.

SUBJECTIVE SYMPTOMS

Murchie, in 1913, writing in the *Glasgow Medical Journal*, concludes that no reliance can be placed on the subjective symptoms, owing to the mental state of the patients with dementia praecox. He finds corneal sensibility diminished, light reaction present, but slow; while the psychic and sensory reflexes were normal in 18 per cent., sluggish in 20 per cent. and absent in

62 per cent. of dementia praecox. The fundus changes observed were venous swelling with tortuosity in some cases, arteries contracted, pallor of disc, congestion of disc and sometimes a mixture of the two; that is, nasal half congested and temporal half pale, which confirms certain of Tyson's descriptions. In other words, he finds a papillitis, which appears in other conditions also, of the same type as in dementia praecox. He goes on to state that a differential diagnosis cannot be made on eye findings alone, but changes in pupils and fundi with vague and mental symptoms might assist in diagnosis.

Cooke of Seattle, writing in *Ophthalmology*, confirms in general Thomas' findings. He examined four groups, and his conclusions are more clear-cut and distinct than those of Thomas. He found, in twenty-four cases of general paresis, twenty-eight cases of dementia praecox, and twelve cases of maniac depressive, the same filling in of the porus opticus in all stages, with pallor of papillomacular bundle, indistinctness of disc margins and vascular abnormalities in all three groups, which rather conclusively contradicts Tyson's statement that these changes are peculiar to dementia praecox alone. Like Thomas, he believes that these cases are practically all syphilitic. He examined the discs of seventeen normal persons, finding some of the above conditions present in four, which leads us still farther away from the idea of a syndrome in dementia praecox.

PATHOGNOMONIC SIGNS

Professor Bumke, in an able article on the pupillary disturbances in dementia praecox, gives us the nearest approach to a syndrome which I have found. He describes (1) a lack of the psychoreflexes; (2) the lack of the normal restlessness of the pupil, and (3) the absence of reflex mydriasis in response to sensory stimuli in the presence of maintained light. In regard to the absence of the normal restlessness of the pupil, he goes on to state that the pupil of a normal person is in a constant state of motion. The loss of this springiness of the pupil, as it might be designated, is invariably indicative of dementia praecox. He says the reason that this has not been observed by investigators in general is that it cannot be determined with the naked eye, he himself having used a suitable magnifier in his investigations. His observations, as just noted, were confirmed by Heubner in a large majority of 236 patients. Sioli also found a lack of springiness of the pupils and the loss of the sensory reaction in twelve out of thirteen hebephrenics, 16 times in 17 catatonics, and 9

times in 10 paranoid dementias. Bumke also goes on to say that the pupils are constantly larger than normal.

PERSONAL OBSERVATIONS

My own observations include an examination of fifty-three cases of dementia praecox and thirteen cases of maniac depressive insanity. The changes in the disc noted in dementia praecox did not confirm Tyson's observations except in part. I did not find one instance of disc alteration as shown in the third illustration of his article. The changes were more than those described by Thomas. The most constant feature of the change in retinal appearance was the dilated and sometimes tortuous condition of the veins, together with the contracted arteries. The disc at times was of a normal appearance, while in others it would range from a hyperemic condition to a quite well-marked papillitis, and occasionally would show a condition described by both Tyson and Thomas, that is, the inner side congested, and the temporal side pale. I found the cupping normal in 40 per cent., diminished in 45 per cent. and filled in 15 per cent.

I attempted to examine the fields; but, naturally, this was impossible, except in a limited number of instances. I did take, however, the form and color fields in fifteen selected patients of dementia praecox in the more recent stages of the disease. Wherever there appeared to be any discrepancy in their answers, I took the fields a number of times, and did not record results unless they were repeatedly corroborated. The charts show, in the majority of instances, the concentrically contracted field for form and colors, with frequent interlacing of the color-fields. The contraction ranges from slightly less than normal down to 30 per cent., which is practically the same result shown in the investigation of the others mentioned. The maniac depressive insanity patients showed no cupping with deep congestion in 35 per cent. (sometimes with slight elevation of disc), and partially filled in the remainder.

In the examination of the pupils, my efforts were directed toward a group of phenomena as described by Bumke. My results largely confirmed his observations. I found, in 65 per cent., a lack of the springy pupil which he described; while the sensory reflex was absent in 70 per cent. In observing these phenomena, I used a convex lens of 13 diopters, since I found, as suggested by him, that it was impossible always to accurately see the movement of the iris when its activity was lowered but not absent. I also found that this lack of springiness in the

pupil, as well as the lack of sensory reflex, was practically always absent in the same case. (Bumke and Weiler never missed these pupillary reactions in sane people—Heubner in three only.) The pupils were larger than normal in nearly all cases.

Of the thirteen cases of maniac depressive insanity, I found the lack of springy pupils and absence of the sensory reflex in 30 per cent. It is possible that the reason my percentages in recording these phenomena are lower than those recorded by Bumke and others quoted by him is because the cases which I examined were of fairly recent origin. And, it is possible that in later stages of the disease the percentage of these changes referred to will be higher.

EYE TESTS WITH ADRENALIN

An interesting contribution to this subject is given by Bayard Holmes of Chicago, in a *Lancet-Clinic* editorial, July 24, 1915. He goes on to state that it has been demonstrated in a large number of cases experimented on that a solution of adrenalin, 1:1000, placed in the normal eye, did not cause mydriasis at the end of thirty minutes. He further gives the results of some observations of J. H. Schultz, and also of Richard Cords of Bonn, in which they found, in cases of indubitable dementia praecox, adrenalin mydriasis came on at the end of ten minutes and persisted for half an hour or longer. He concludes the editorial in the following words:

"The value of this reaction as a means of differential diagnosis may not be great, but its usefulness in suggesting the direction of research can hardly be overestimated. When we take into consideration the fact that the blood pressure in dementia praecox is always low and that subcutaneous injections of adrenalin do not raise it; when we notice that the Abderhalden reaction in this disease indicates a dysfunction of the glands of internal secretion, especially of the sex glands; when we find that morphologically the sex glands and most of the other glands of internal secretion are obviously dystrophic, as shown by modern microscopical methods, when we take into account the secondary origin of most pluriglandular disturbances that we know anything about, such as hypo- and hyperthyroidism and pancreatitis—then we begin to value the disturbances of the glands of internal secretion as promising ultimately a way to discover the infection on which dementia praecox depends."

In conclusion, the majority of observers so far, myself included, do not find disc and fundus changes that are pathognomonic of dementia praecox. I believe that the researches of Bumke point more clearly to the possibility of an eye syndrome

in this disease; i. e., the absence of psychic and sensory reflexes, the natural "springiness" of the pupil, and the enlargement of the pupil. The classification of the different forms of insanity is not always an easy thing, and it is possible that the discrepancy noted between the results of the different observers may be due to the fact that our present classification of dementia praecox is not quite as it should be.

If future investigators will follow up the suggestions of Bumke in regard to pupillary phenomena, and also the hint given us by Holmes, it may lead to something definite in the formulation of an eye syndrome in dementia praecox.

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DISCUSSION

DR. D. T. VAIL, Cincinnati: During the past year I have been busily engaged in preparing an article for Casey Wood's Encyclopedia of Ophthalmology on the subject "The Pupil in Health and Disease." I made a thorough investigation of the literature on the pupil in dementia praecox and found it was the consensus of opinion, that there are no pupillary signs, that is, none pathognomonic of this disease. In fact there are no invariable and constant pupillary signs of any disease in the body. While many cases of dementia praecox presented the pupillary phenomena described by the essayist there were many cases of other diseases which also showed the same kind of pupils.

Dr. Langdon, a distinguished neurologist of Cincinnati, presented a paper years ago in which I believe he made the claim that he had found the colon bacillus in nearly every case; this he concluded to be the cause of dementia praecox.

DR. GEO. A. KEIPER, Lafayette, Ind.: Dr. Teal has quoted Dr. Holmes in this matter, and it might be of interest to know what his last contribution is. At the last meeting of the American Academy of Railway Surgeons he has decided, and has evidently proved his contention, that this disease is caused by intestinal putrefaction, especially in the cecum, and recommends operative interference, confirming what Dr. Vail says. It seems to have its origin in the cecum.

DR. KIRKENDAHLE: I have had experience in these cases for twenty years and have watched them with much interest, thinking we might be able to find these changes, but I have as yet been unable to see changes in the eye that could be classed as cause and effect. I have found the patient's breath is always foul, and it is my opinion, and I believe it will be developed, that you will find a serum that will relieve it some day.

DR. F. PARK LEWIS, Buffalo: Dr. Keiper, quoting Holmes, says it is found in the cecum. Dr. Lane also believes that true of the colon bacilli. We get the ductless gland infection and it makes a vicious circle.

A NEW MAGNET

WALTER B. LANCASTER, M.D.

BOSTON

The object of this is to furnish as large and powerful a magnet as possible that is still portable. A number of these have been made for the base hospitals. It would be better to have a larger one, but then it would not be portable. This is as large as it seems possible to make them and have them portable. It weighs about 50 pounds. It will pull as much at 7 or 8 mm. as the Haab at 10 mm. You can get a little nearer the eye with this because it is easier to manipulate so that in effect it is almost up to the Haab in power. With this it is comparatively easy to approach the eye and manipulate the pole.

This second tip was proposed by Dr. Greenwood for use in brain surgery, and it can be introduced into the brain to remove pieces of shrapnel, etc.

This magnet can be used with an automobile battery. It is made in Boston by a mechanic named Gleeson. This magnet cost \$125, a small one costs \$25, and a large one \$350. It can be used without a foot switch but the better practice is to use a foot switch.

COMMITTEE REPORTS

REPORT FOR THE AMERICAN BOARD FOR OPHTHALMIC EXAMINATIONS

Immediately following the last annual meeting of the Academy, the first examination of candidates was held by the Board at Memphis, Tenn., Dec. 14, 1916. A second examination was held in New York City, June 7-8, and a third examination at the Singer Memorial Research Laboratory, Pittsburgh, Oct. 27, 1917. At these examinations 120 have been awarded the certificate of the Board.

In addition, 29 applications have been received, of which 11 have been refused, and 18 are still pending before the Board, the applicants having failed to appear for written and practical examinations, or not having submitted in time the required case records, or action has been deferred for further investigation, or other reasons.

After careful consideration, the Board decided to require that every applicant for certification should sign the following:

"I hereby pledge myself not to accept commissions from any optician for work sent to such optician, nor to divide fees, nor to pay commissions to any persons referring cases to me."

Failure to observe this condition, on which all certificates are granted, is an essential fraud, which justified the revocation of the certificate. It is expected that those who cannot honestly give this pledge and live up to it, will abstain from applying for examination.

Shortly after the last annual meeting of the Academy, and the first examination of the Board, we were deprived of the most valuable assistance of one of the Academy representatives—our active, energetic, enthusiastic colleague—Dr. Wendell Reber, whose untimely death we cannot cease to regret. To fill temporarily the vacancy thus created, the president of the Academy appointed Dr. William H. Wilder of Chicago, whose acquaintance with the work already done was of especial advantage in this emergency.

While the work of the Board has been rendered more difficult and less productive by reason of the existing war (four members of the Board being now on active military duty), we

feel that much has been done toward the attainment of the object aimed at. More still may be expected when full cooperation has been effected with the American College of Surgeons.

As stated in the reports submitted to the recent meetings of the American Ophthalmological Society and the Section on Ophthalmology of the American Medical Association, the American College of Surgeons has already taken action constituting this Board the Ophthalmic Credentials Committee of the College. It is recommended that in accordance with this arrangement the Academy nominate its present and prospective members of this Board for members of that Ophthalmic Credentials Committee, as has already been done by the American Ophthalmological Society and by the section of Ophthalmology of the American Medical Association.*

EDWARD JACKSON, *Chairman.*

The general committee representing The American Laryngological, The American Otological, The American Laryngological, Rhinological and Otological, and The Academy of Ophthalmology and Oto-Laryngology has had several meetings during the past year.

The committee recommends the obtaining of a special degree from the post graduate department of leading universities by all those who desire to enter the specialty of oto-laryngology.

This degree is to be obtained after examination conducted by the University.

It will be open to all:

1. Who have acted as interns in a standard hospital in either medicine or surgery, or both, or have passed a term of years in practice.
2. Who have also subsequently served as intern in the oto-laryngological department of a standard hospital for the period of eighteen months.
3. Who have subsequently pursued a curriculum of study as laid down by the University in which the candidate is seeking his post graduate degree. This course to be of not less than six months.

Dr. Wishart, chairman of the general committee, also recommends that the committee in each society be continued without change in order that the men who are familiar with the work may go on with it.

J. M. INGERSOLL.

JOSEPH C. BECK.

THOS. E. CARMODY.

Committee.

*Note.—Committee continued and to report after the close of the war.

REPORT OF COMMISSION ON THE ETIOLOGY OF IRITIS

Two years ago a paper was presented to the Academy entitled "The Etiology of Iritis as Determined by Laboratory Methods." In it the author expressed his "profound dissatisfaction" with prevailing views of the causes of iritis and presented careful studies, clinical and laboratory, of a series of cases, in which the possibility of new etiologic factors was suggested. It seemed to him that the time had come for a resurvey of this problem. Let us quote his closing words:

"What the ophthalmic profession just now needs in the domain of the uveal tract is not more treatment, but *more light* which we fervently hope may lead us to better and more accurate treatment of this malady that is fraught with such grave consequences to the precious function of sight."

Many of us will recall the slight figure, vibrant with nervous energy, the expressive face, and the high-pitched earnest voice with which the author uttered his plea for *more light*. More Light! These words perfectly express the life-long yearning of Wendell Reber. Always he sought to illumine the dark paths of ophthalmology, and his infectious enthusiasm stirred in many others the desire for "more light."

Immediately on the conclusion of his paper, a motion was passed creating a "Commission to Investigate the Etiology of Iritis." Dr. Reber was, of course, made chairman, with Drs. W. B. Lancaster and John Green, Jr., associates.

With characteristic thoroughness, Dr. Reber prepared an elaborate scheme for the guidance of our collaborators, enlisted the services of a number of laboratory workers in Philadelphia and elsewhere, and secured promises of cooperation from ophthalmologists in many parts of the country. The fruits of this preliminary work were presented to the Academy at the Memphis meeting. Then came the wholly unexpected illness and death of our leader.

The work of the commission, so auspiciously begun, came to a standstill. Neither of the surviving members found it possible to assume the responsibilities of chairman, and several months elapsed before the third member, Dr. Walter R. Parker of Detroit, was appointed to fill the vacancy caused by Dr. Reber's death. He, too, found it impossible to accept the chairmanship.

Late in July, Dr. Green consented to act as temporary chairman up to the time of this meeting.

It may well be doubted whether, even under favorable circumstances, we should have made much progress this year. Since April 6, the energy of every medical man has been bent toward helping to make the world "safe for democracy": We have been too intimately concerned with our share in the mighty struggle to give much thought to problems of purely scientific interest. If we are "marking time" now, we shall be ready to go forward at the first opportunity.

It may not be out of place to present a brief résumé of some of the more important papers appearing during the year on the etiology of iritis.

(1) De Schweinitz and How (Trans. Coll. of Physicians, Phil. Vol. 38 P. 337) record the case of a 5 year old boy with polyarthritis (Wassermann and tuberculin reactions, negative), who developed an iritis in the right eye and a kerato-iridocyclitis in the left. The disease in the left eye proved intractable, an exudate formed in the anterior chamber and the globe had to be removed.

(2) A boy of 13 with a polyarthritis of undetermined origin came under Langdon's observation. (Trans. Coll. Physicians, Phila. Vol. 38, P. 356.) A small gray node appeared on the pupillary margin of the iris. This slowly increased in size, accompanied by the formation of a gray exudate. Tuberculin reactions were negative, both as to the eye and generally. The eye finally became blind.

(3) Morax (*Ann. d'Oculistique*, Vol. 154, P. 45) observed repeated attacks of iritis in an officer with acute dysentery. The iritis was a late manifestation and came subsequent to an attack of arthritis in which several joints were involved.

(4) The rôle of dental foci in the production of metastatic eye infections has been investigated by Levy, Steinbugler and Pease (*Jour. Am. Med. Assn.*, Vol. 69, No. 3, P. 194). Fifty-seven patients with various eye conditions presented dental foci of infection—pyorrhea, apical abscess, etc. Appropriate dental treatment cured or benefitted twenty-nine. Of the fifty-seven there were twenty-nine cases in which the iris was involved, either alone or in conjunction with other structures. Of these 4 were cured (13.7 per cent.), 13 were improved (44.8 per cent.) and 12 were not benefitted (41.5 per cent.). One instructive case was that of a woman, age 30, (Wassermann + + + +) with double

iridocyclitis, resistant to local treatment and mercury. Roentgenograms revealed apical infection of seven teeth. Extraction of all was followed by immediate relief from pain and very prompt recovery.

(5) In two hundred cases of iritis occurring in private practice Lang (Trans. Royal Acad. of Medicine, July, 1917), found the etiology as follows: syphilis 6 per cent., gonorrhea 12 per cent., tubercle 11 per cent., general affections 8.5 per cent., local affections (septic foci in the skin or mucosa) 25.5 per cent., and pyorrhea 37 per cent. In discussion Leslie Paton reported a case of iritis with corneal abscess and hypopyon in which cultures from both yielded staphylococcus aureus and micrococcus tetragenus, the last named practically never found except in abscesses about the teeth, nasopharynx or lungs. A septic tooth was the only source of infection.

(6) The association of iridocyclitis with Mikulicz disease has been noted by Giessing (Abst. *Berl. klin. Wchnschr.*, Vol. 53, P. 30); with bubonic plague by Zimmer (*Clin. Opht.*, Vol. 22, P. 30); with tonsillitis by Sobotky (*Boston Med. and Surg. Jour.*, Vol. 196, P. 806). Conjunctivitis and iritis following an injury to the eye with the acrid juice of *Chelidonium majus* is reported by Hilbert (*Centralbl., f. Prakt. Augenheilk.*, Sept. and Oct. 1916).

(7) In 1915, Rosenow had shown that intravenous injections in rabbits of streptococci from various lesions in man and animals produced iridocyclitis, this uveal localization being due to peculiarities in the organisms. Irons, Brown and Nadler (*Jour. Infect. Dis.*, Vol. 18, P. 315) experimented "with the idea of tracing the changes in the power of an organism (streptococcus) which had already given rise to iridocyclitis in a patient to produce similar lesions in animals after varying periods of residence in the original host, of residence in animal tissue and of growth on culture media."

A patient suffering from chronic dacryocystitis developed iridocyclitis. Hemolytic streptococci isolated from the tear sac produced typical iridocyclitis in three out of four rabbits injected intravenously. Hemolytic streptococci isolated one month later produced iridocyclitis in two of four rabbits injected. Subsequent cultures failed to produce iridocyclitis in five series of rabbits, due to some change in the invasive power of the organism for ocular tissues. Attempts to obtain a return of invasive power for tis-

sues of the eye of streptococci, which had lost it, by growing the organisms in the living eye, were unsuccessful.

The above authors conclude:

"These experiments seem to indicate that the invasive power of an organism for special tissue may change within a short period of time during residence in the original host, during animal passage, and in culture without pronounced or constant changes in cultural characteristics, or in general virulence for animals."

JOHN GREEN, JR.

WALTER B. LANCASTER.

Commission.

TRANSACTIONS

OF THE

TWENTY-SECOND ANNUAL MEETING

OF THE

American
Academy of Ophthalmology
and Oto-Laryngology

OTO-LARYNGOLOGICAL DIVISION

HELD AT
PITTSBURGH, PA.
OCTOBER 29 AND 30, 1917

THE ROUTINE OTO-LARYNGOLOGICAL EXAMINATION OF FOUR THOUSAND APPLICANTS FOR THE AVIATION SECTION OF THE SIGNAL CORPS OF THE UNITED STATES ARMY

WITH TOTALS AND AVERAGES OF THE VESTIBULAR ROTATION REACTIONS OF THE FIRST TWO THOUSAND

AUSTIN A. HAYDEN, M.D.; F.A.C.S.

CHICAGO

THE WAR AND THE AVIATORS

Our country is engaged in a world war of titanic proportions. Those most familiar with military affairs, believe that the deciding factor may easily be the "armies of the air"—that our own aviators may win for us and our allies a decisive victory over military autocracy with lasting peace and happiness for all the peoples of the world.

Our government has called for thousands of men for the aviation section of the signal corps. The tens of thousands who responded have been examined under the general direction of Lieut.-Col. T. C. Lyster, M.C., and the particular supervision of Majors Isaac Jones, M.R.C., and Eugene Lewis, M.R.C.

THE OTO-LARYNGOLOGICAL WORK OF THE UNIT AND THE SCOPE OF THIS PAPER

Luckily, last winter the author took the equilibration work offered by Jones at Pennsylvania, and was consequently able, as a volunteer worker, to help Major Norval H. Pierce, M.R.C., establish the physical examining unit at the Illinois Charitable Eye and Ear Infirmary last June. Over 4,000 applicants have been examined.

The oto-laryngological findings of this series are reported for a twofold purpose:

- (1) To give those, who expect to do this work, some of the more important technical details that have been elaborated and which have enabled us to carefully examine as many as sixty applicants in a single day.

(2) To further stimulate interest in the functional tests of the semicircular canals. This field, though old, had never been widely explored until the war created a national demand for the work Jones had fortunately systematized and put to practical use.

It is hoped that a sufficiently large number of cases will soon have been studied by various men, so that the clinical interpreta-



Fig. 1.—Lined up for the whisper test.

A. Pile of Records ready to be marked; B, Assistant stopping up the candidate's left ear with his left index finger—and then the right; C, The last man to pass by the examiner. His record was consequently on the top of the pile, and his hearing was taken first but he will be the last of the line as they march back for the examiner to return the papers; D, The first man to hand his paper to the examiner. His record, consequently was at the bottom of the pile, and his hearing will be taken last, but he will head the line as it marches back for the papers. This arrangement, as well as that shown in Figure 2, saves much time. Thirty to forty men can easily be handled in this way.

tion of the findings can be as clear as a temperature chart or a blood-pressure reading.

THE APPLICANTS AND THEIR GENERAL ATTITUDE

They have been almost without exception of the very finest type of red-blooded young American manhood imaginable. The extensive publicity that was accorded the severity of the physical examination apparently kept all but the almost physically perfect away. The percentage of rejections has consequently been very small.

Without exception every man has literally been crazy to fly.

Tears have not infrequently been shed when a boy was rejected. The airship seems to be the most immediate means of "getting the Kaiser." For that reason there is no malingering to escape service. Many have tried to "get by" with disqualifying defects.



Fig. 2.—Lined up for the watch test.

Each man carries his own record, on which the examiner marks the findings as soon as the test is made. *A*, clerk stopping up right ear (in which hearing has already been taken); *B*, examiner's hand screening eyes so that patient cannot see where the watch is; *C*, watch in position. Examiner manipulates it at various distances and in different positions; sometimes with his hand open, and sometimes, closed tightly over the watch; to obtain correct answers to the order, "Tell when as soon as you can hear the watch tick." (The same watch is used all the time.); *D*, record spring-clip boards which Major Casey A. Wood, M.R.C., generously furnished when the work began, and which have been extremely useful.

Strict silence is, of course, maintained during the whisper and watch tests. The men are even cautioned against moving their feet. Both assistant and examiner should work in their shirt sleeves as the movement of their arms in their coat sleeves makes enough noise to be troublesome.

ROUTINE EAR, NOSE AND THROAT EXAMINATION

This embraces the following:

- (1) History of ear, nose and throat trouble including head injuries and dizziness from any cause.
- (2) Examination of the nose, throat, nasopharynx, external auditory canal, tympanic membranes, and cochlea.
- (3) Testing the hearing by whisper and watch.
- (4) Semicircular canals: Equilibration by rotation tests for nystagmus and caloric tests for vertigo as objectivized by past-pointing and falling.

History.—The candidate is most carefully questioned regard-

ing otalgia and otorrhea, tinnitus, head colds and sore throat, head injuries and attacks of sea-sickness or other vertigo from any cause whatsoever. Among these are mentioned riding on trains, merry-go-rounds, automobiles, horses, elevators; looking from the roofs of high buildings; sitting in swings, hammocks



Fig. 3.—Rotation of eyes to extreme right to bring out latent spontaneous nystagmus.

The candidate has fixed on the red water pipe a block away, and has been told to look steadily at it. The chair has been turned far to the left, so that his eyes are looking to the extreme right. Jones says that a horizontal nystagmus in this position, if attended by a nystagmus of similar character and degree in Figure 4 is not abnormal.

and rocking-chairs; or dizziness from looking at any of these things.

Two men presented very interesting histories. They were so dizzy the first (and last) times they rode horse-back that they

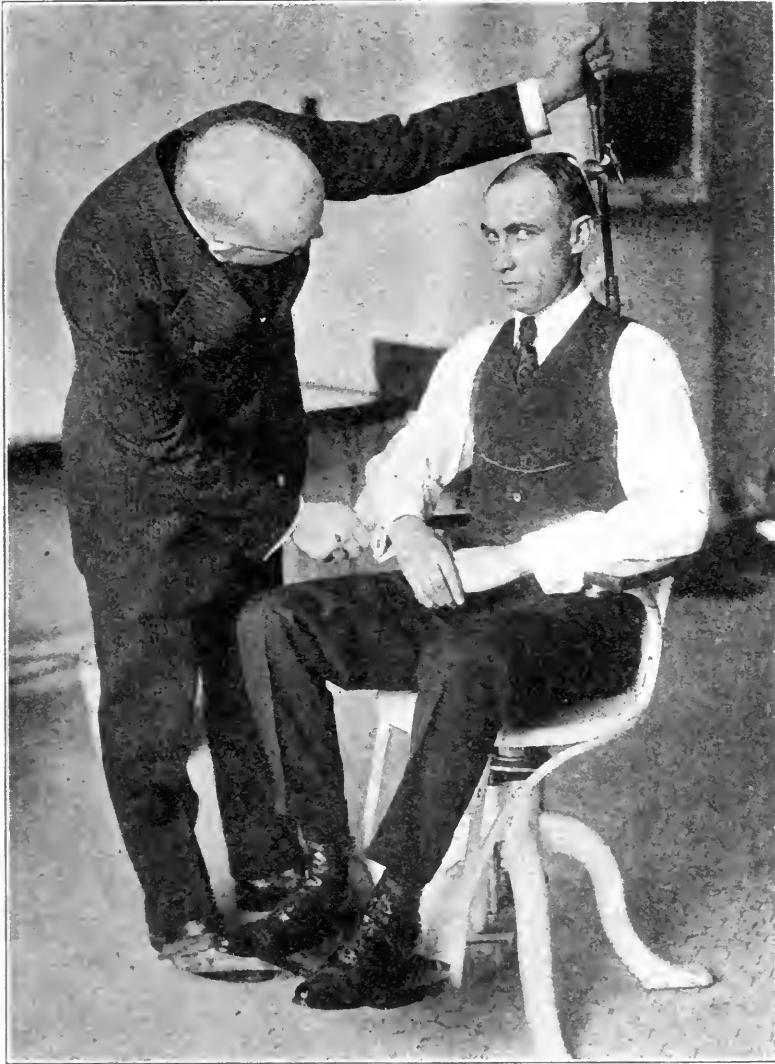


Fig. 4.—Same as Figure 3, but with eyes rotated to extreme left to bring out latent spontaneous nystagmus.

fell from the saddle, one with a fractured skull; the other sustaining an extensive scalp and forehead wound. They showed

extreme hypersensibility to turning. Both were, of course, disqualified.

A physician of the author's acquaintance is made so dizzy by looking at a person rocking in a chair that he becomes nauseated.

Another physician whom the author induced to try the chair



Fig. 5.—Extreme elevation of eyes to bring out latent spontaneous nystagmus. In this position, one patient has been seen with a vertical nystagmus upward. Unfortunately he refused further examination, after having been rejected for service. (Drawing the lower lids down gives the surgeon a much better view of the eyeball.)

the first day it came, developed a nausea, without actual vomiting, that lasted for twelve hours after only five slow rotations had been made.

Examination of the Nose.—Serviceable breathing and clean, healthy noses are required. For that reason, ozena, involvement of the sinuses and obstructive deflections of the septum reject the applicant until the condition has been cleared up. These conditions hold up from 5 to 10 per cent. of each day's class.

Throat.—The old mooted question as to what constitutes a normal tonsil came up with almost the first man to be examined. For the practical purpose of this work it was decided to require tonsillectomies of three classes of men:

(1) Those who had had recurrent tonsillar infections as often as once a year.

(2) Those whose tonsils were obstructive.

(3) Those from whose tonsils free pus could be expressed in considerable quantities.

On this basis about 5 per cent. of the men have been operated on.

Nasopharynx.—This is routinely examined by the excellent method initiated by Major Norval H. Pierce, using the Michel mirror. So far, no adenoid masses of anything like obstructive proportions have been found.

External Auditory Canal.—Aside from a goodly number of impacted cerumen cases and a few boxer's ears, some of which had been traumatized to such an extent that the canal was barely patent, no pathology has been seen here.

Tympanic Membranes.—Inasmuch as perforations positively exclude from the service, the drums were most carefully examined, and in doubtful cases, probed and catheterized for the perforation sound. About one-half per cent. have been excluded on this account. One of these cases was of particular interest: Dr. William H. Ballenger had performed his mastoid operation on the right ear some five years previously. The hearing was perfect and the drum completely intact. The passage from the external auditory canal back into the antrum could be probed, however, to rather an unusual depth and Dr. Lewy was able to produce the fistula test.

Cochlea.—The hearing is taken by the whisper and by the watch.

Whisper: The men are lined up and marched single file past

the examiner who collects their papers and places them on his table at one end of the room in their exact order (Fig. 1). The line stands facing the wall 20 feet away. The clerk stops up the left ear of the last man, with his left index finger and then the right with his right index finger. High and low pitched numbers and words are whispered in the usual way. The findings are immediately recorded. Each man's paper is returned to him as he marches by the examiner again. Full 20/20 for high and low tones is required.

Watch: A line is formed in the center of the room (Fig. 2). The clerk stands behind the man being examined, to stop up first the right, and then the left ear, while the examiner stands in front screening the eyes with one hand and manipulating the watch with the other. The results in fractions of forty are immediately noted on the record which each candidate now carries. 40/40 is considered normal, although a considerably smaller fraction is possible if the whisper test showed full 20/20.

Semicircular Canals.—Rotation and caloric tests are used to stimulate the horizontal canal working alone, and the vertical canals, working together. The reactions manifest themselves in (1) nystagmus and (2) vertigo.

NYSTAGMUS.—This is a rhythmic to-and-fro movement of the eyes made up of a slow component, the vestibular pull, and a quick component, the cortical jerk. As the latter is the more noticeable, it designates the direction of the nystagmus which is, consequently, always opposite to the endolymph movement.

Spontaneous Nystagmus: This is looked for first. The candidate fixes on a distant object straight ahead of him. To detect any lateral movements, Major Jones then turns the chair to the right (Fig. 3), and then to the left (Fig. 4), until the object is almost out of view. For extreme elevation of the eyes (Fig. 5), the examiner's left index finger is fixed on as he moves it upwards, and for extreme depression (Fig. 6) his right, while the lower lids are pulled down in the first instance, and the upper are held up against the brow in the second.

Horizontal nystagmus under these circumstances is frequently seen; rotary, occasionally. Jones considers vertical nystagmus pathognomonic of a brain stem lesion.

Induced Nystagmus: The candidate is seated in the Jones chair (Fig. 7) with his head inclined 30° forward and rotated with his eyes closed, ten times to the right, in exactly twenty

seconds. He is now directed to look at the distant object. We use for convenience a red water pipe on a building across the street. This is slow turning. As a variation of a few seconds will cause a marked difference in the duration of the nystagmus, the examiner should always time himself with a stop watch. The duration of the horizontal nystagmus to the left, which is thus produced by the stimulation of the right and left horizontal canals, is noted



Fig. 6.—Extreme depression of eyes to bring out latent spontaneous nystagmus. Holding the upper lids against the brow gives the examiner a much better opportunity to observe the ocular movements.

and recorded. Twenty-five seconds is considered normal. A variation of ten either way is passable. Our own series, however, shows a considerably shorter period to be the average.

The candidate is next rotated to the left in the same manner, producing horizontal nystagmus to the right.

Exact Duration: The duration of the nystagmus is frequently difficult to determine with precision. To do this, the



Fig. 7.—Turning to produce nystagmus, right and left, from stimulation of horizontal canals.

Candidate's head 30° forward in head-rest; eyes closed; hands grasping arm of chair; feet firmly on foot-board, with heels pressed tightly back against chair; examiner turning chair steadily by its handle and timing the revolutions.

examiner should pull the lower lid down and away from the eyeball with his right hand, and at very close range continuously watch some particular vessel on the sclera in its relation to a



Fig. 8.—Testing for spontaneous past-pointing.

Here the examiner can hold the chair perfectly still with his foot, as its blind-folded occupant is not dizzy and sits perfectly quiet. This same arrangement was used on the old chair (Fig. 12, left chair) which was pressed into service. To hold it still, the examiner simply pressed the heel of his shoe tightly against the foot-rail, allowing the ball of his foot to protrude through to rest firmly on one of the pedestal's legs. The examiner's right index finger is steadied by his left hand, both pinioned firmly against his body. The candidate's arm is fully extended and stiff. He is ready to raise it straight and bring it straight down back to touch the observer's finger. It will assume the same position, of course, when the past-pointing stops.

fixed point on the lid margin. The intelligent candidate, for the reason that his own retina is the most sensitive index of his ocular movements, can render valuable assistance, especially when the excursions are small, if he will announce when the water pipe stops moving. This statement must always be verified carefully by the examiner's own observation, as often the



Fig. 9.—Starting the chair for the fast-turning for past-pointing ten times in ten seconds.

Applicant's hands are holding on to arms of chair tightly, his feet firmly placed on the foot-step, with his heels well back against the chair. The examiner's right hand is starting the rotation by pulling the arm-rest around so that the upright which he is holding in his left hand and with which the rotation is continued, will be saved the heavy strain of starting.

inattentive or overanxious will volunteer this information when the nystagmus is still plainly visible. By following these simple precautions the use of convex lenses to relax the accommodation and magnify the movements has not been found necessary.

The nystagmus spoken of in this paper is after-nystagmus of the second degree. No after-after-nystagmus has been seen. Major Norval H. Pierce made the very interesting observation that nystagmus stops with vomiting. In douching etherized



Fig. 10.—Stopping the chair after the fast-turning for past-pointing.

The last of the tenth revolution is just being completed. The examiner's right foot is pressing down on the inner end of the foot pedal releasing the spring to stop the chair. The outer end of this same pedal, when pressed down, sets the spring so that the chair will revolve. His right hand is stopping the chair to save the foot pedal as much as possible.

patients' ears, the author has seen the ocular deviation disappear when emesis occurred.

REST PERIOD. TWO CHAIRS. NAUSEA.

By allowing an interval of two or three minutes to elapse before making the tests for vertigo, the liability to vomiting has been decreased from 16 per cent. in the early work of the unit to less than 2 per cent. at the present time. This is extremely

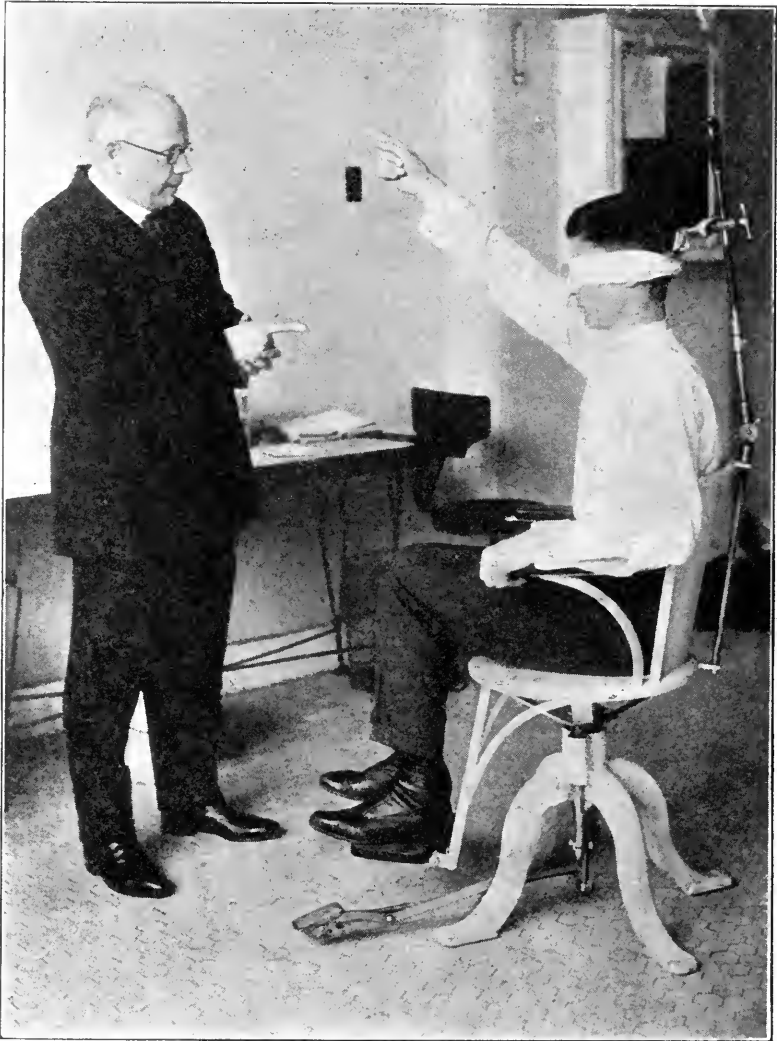


Fig. 11.—Measuring the past-pointing.

The chair is locked. The right arm is pointed first, then the left and so on until each touches the examiner's finger.

desirable as the nausea is often distressing and prolonged. Some men complained of having vomited as many as twenty times and of being nauseated well into the next day.

Two chairs can be used to great advantage (Fig. 12). The first man can rest while the second man's nystagmus is being taken. Then the first is ready for the turning for vertigo.

The regulation Jones' chairs have been in such great demand that it was necessary to use an older make that had no locking



Fig. 12.—Arrangement of room to prevent men from forming preconceived ideas as to how they should react to the tests.

Candidates seated on bench looking away from chairs out of the window. Examiner and clerk guard against peeking. In the early work they were screened off in one corner of the room. This soon grew so noisy that finally the present plan was evolved which has proved satisfactory. The Jones chair is shown to the reader's right, the old chair to the left, with the bucket close at hand for emergencies. The vomiting is now less than 2 per cent.

device. This chair can be used with satisfaction, if the examiner will place the heel of his shoe firmly against the foot-rail, and the ball of his foot on the pedestal of the chair (Figs. 8 and 12).

The danger sign of oncoming nausea is a cold clammy sweat, rapidly followed by intense ashy pallor.

VERTIGO.—Vertigo is objectivized as past-pointing and falling.

Past-Pointing: Here the blindfold is of great assistance, and is routinely used, as there is an almost irresistible tendency to look to see where the examiner's finger is.

Spontaneous Past-Pointing: This is always looked for first. The candidate is directed, "Point your right index finger straight

out and touch my finger." (Fig. 8.) "Raise your arm straight up. Straight back down to touch my finger." (Fig. 9.) Then the left arm is raised and lowered. Each arm should describe an arc of something less than 90 degrees. To prevent movement of his own finger, the examiner should always steady his hand against his body as shown in Figs. 8 and 9.

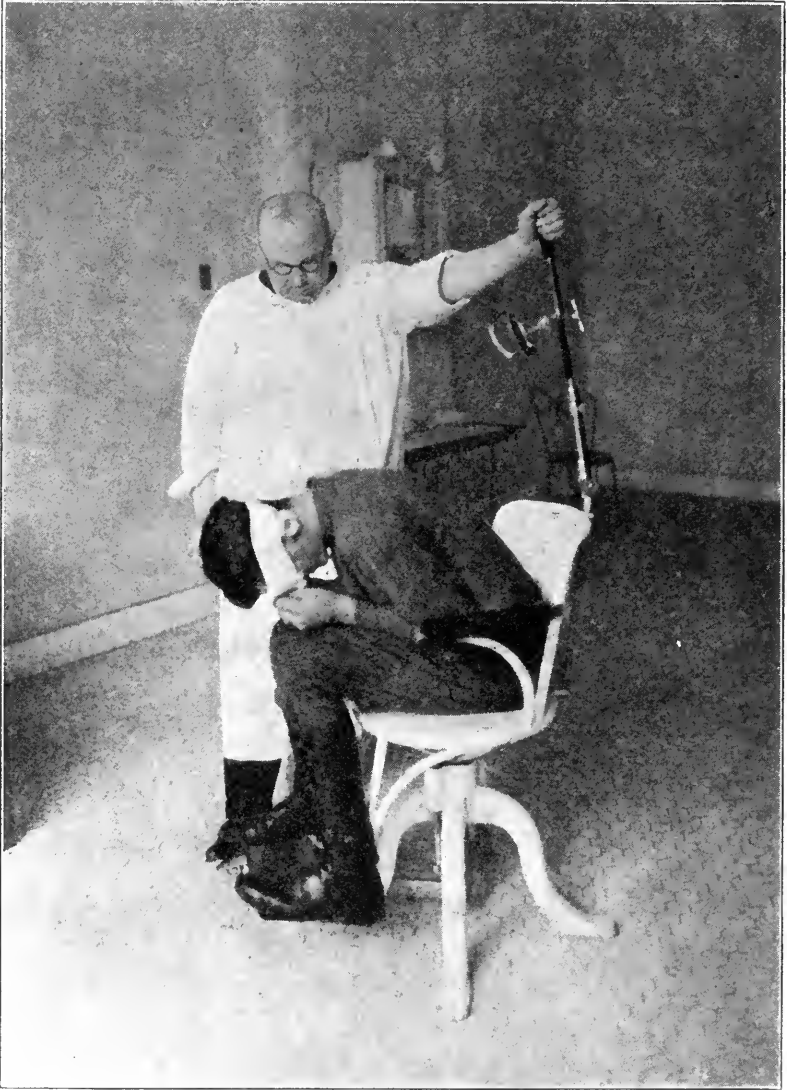


Fig. 13.—The slow turning (five times in ten seconds) for the falling reaction.

"Knees together—Fists on your knees—Heads down on your fists," brings the man into this position where his vertical canals are level with the floor, so that their endolymphs will set in motion by the turning.

Induced Past-Pointing:

(a) *Position.* The head is inclined 30 degrees forward in the head-rest, so that the horizontal canal will be exactly in the horizontal plane. The candidate is told to hold on to the arms of the chair tightly, and to keep his feet on the foot-step with heels firmly pressed against the chair. If these precautions are disregarded, the examiner will often be struck by a flying arm, or painfully kicked by the boot of a careless applicant.



Fig. 14.—A normal falling reaction after turning to the right.

(b) *Starting, turning and stopping chairs.* Rotation is routinely to the right first, and then to the left. The examiner takes hold of the arm of the chair and quickly and forcibly pulls it to the right, continuing the rotation with his other hand on the upright handle (Fig. 9).

Ten turns must be made in ten seconds—this is just about as fast as the chair will go steadily. Until he has turned several hundred, the examiner should be timed by a third person, as the rotation is too rapid to allow him to hold the watch himself.

In the last half of the tenth revolution the chair should be slowed down so that the minimum strain is put upon the foot pedal bar when its lock is released (Fig. 10). Care in starting and stopping, make adjustments of the chair seldom necessary.

(c) *Counting and recording past-pointing.* Immediately when the chair stops locked (Fig. 11), the procedure to be followed is the same as described above under Spontaneous Past-Pointing.

Normally, after rotation to the right, the candidate will bring his finger down to his (the candidate's) right side of the examiner's finger, and to the left, after turning to the left. This is technically known as "Past-pointing." Normally it is always in the same direction as the endolymph movement.

For the sake of uniformity, the right arm is pointed and recorded first, the left, second, and so on alternately, in this order until each touches the examiner's finger. The same arm is never pointed twice consecutively. Thus the equation 6:1 means that the right arm pointed past the examiner's finger six times, touching it on the seventh attempt. The left arm pointed past the finger only once and touched it the second time, continuing to do so through five more pointings, till the candidate finally succeeded in bringing his right index finger to touch also. Three past-pointings with each arm is considered normal, although this series shows a somewhat smaller number as the average. Other things being normal, one pointing of good proportion and under proper conditions with each arm in each direction will pass. Failure to past point or past-pointing in the wrong direction with either arm calls for the caloric tests. If the responses to these are abnormal, the candidate is rejected.

SOURCES OF ERROR

(1) *Past Pointings.*—If the arms are raised and lowered rapidly enough, a normal individual can almost, if not completely, suppress his past-pointing. Slow movements, on the

other hand, exaggerate it. Each past-pointing pointing should consume about three seconds—one and one-half each for elevating and lowering the arm respectively. Doubtful cases should be done very slowly.

(2) *Cross-Cutting*.—Not going straight up and straight down, waving the hand and groping around in a vain effort to find the finger, must be absolutely prohibited.

(3) *Preconceived Ideas*.—Conceptions as to just how the pointing should be done cause annoying, time-consuming delays. For that reason the candidates are not allowed to witness the tests before going into the chair themselves (Fig. 12).

(4) *Prolonged Turning*.—This should be avoided, not merely because of the nausea and vomiting it is likely to produce, but for the further reason that overstimulation of a sensitive end-organ, like the semicircular canals, is apt to produce abnormal uncoordinated reactions.

(5) *Strict Silence and Rigid Discipline*.—These aid greatly in doing rapid, accurate work. Under these conditions, and with the help of a well-trained clerk, ten to twelve can be carefully examined in an hour.

THE FALLING REACTION

The order, "Knees together—Fists up on knees—Head down on fists" brings the blindfolded applicant into such a position that his vertical canals are in the horizontal plane, where their endolymph will be set in motion when the chair is revolved (Fig. 13). The use of the forward head-rest is unnecessary.

Rotation to the right, five times in ten seconds (this is slow, being only half as fast as for past-pointing) is timed with the stop watch. The applicant when told to slowly sit straight up, will veer markedly to the right (Fig. 14). Turning is now made to the left in the same manner.

Occasionally an exaggerated reaction occurs where the body sways far to the right or left, sometimes with violent struggling to overcome the sensation of falling out of the chair. This stops instantly with the removal of the blindfold.

THE FIRST 2,000 VESTIBULAR REACTIONS TOTALLED AND AVERAGED

Nystagmus.—After turning to the *right*, the 2,000 cases showed an aggregate of 43,749.5 seconds of nystagmus, representing an average of 21.8745 seconds each.

After turning to the *left*, the aggregate was slightly larger, being 44,172 seconds, or an average of 22.086 seconds each.

Past-Pointings.—After rotation to the *right*: The right arm past-pointed 3,853 times, giving an average of 1.9265. The left arm past-pointed 3,723 times, with an average of 1.8615.

After rotation to the *left*: The right arm past-pointed 4,450 times, making an average of 2.225. The left arm past-pointed 3,931 times, with an average of 1.9655.

Of the 2,000 applicants, only 63 were rejected for abnormalities of the kinetic static sense. Of these 52 failed in past-pointing, 7 in nystagmus, and 4 in falling.

Of the total number examined, 355 were rejected as being unfit to become flyers, while 1,645 were pronounced about 100 per cent. physically perfect, by the examining unit.

CONCLUSION

This report represents a large amount of work, and the author wishes to express his sincerely grateful appreciation to those who have so cheerfully and whole-heartedly helped in its accomplishment. Without Dr. A. F. Lewy, the thing could not have been done. Drs. Speice, Birmingham, Smith (the Superintendent of the Infirmary), and Dr. Martin of Lead, S. D., have all rendered invaluable assistance. The entire corps of physicians, nurses and attendants of the Infirmary have been most cordial; and last, but, by no means, least, Captain Lane, M.R.C., who is in charge of the unit, has been most solicitous for the interests of its oto-laryngological department. The caloric tests will form the subject matter of a subsequent paper in the near future.

CAREFUL TECHNIC IN MAKING THE LABYRINTHINE TESTS

GEORGE W. MACKENZIE, M.D.

PHILADELPHIA

In 1907 Bárány published his monograph "Physiologie und Pathologie des Bogengang-Apparates beim Menschen." At about this same time he began to teach the subject to post-graduate students. Many things he then taught were not entirely new; however, to him is due the credit for having developed the subject to that extent which made it possible for one to diagnose quite accurately pathologic conditions of the inner ear by clinical tests.

Bárány occupied a rather favorable position for the study of the physiology and pathology of the inner ear in that he was surrounded with an environment calculated to inspire, with material in abundance, and with excellent critics within easy reach.

Alexander, his senior and earlier adviser, as well as Kreidl, the physiologist, had done considerable investigation along the same line before him. It was Alexander who prompted, if not actually started Bárány in this special line of work, as the earlier publications attest (the two names appearing conjointly). So much for the inspiration.

The material in the Politzer clinic was perhaps as abundant as any that could be found in Europe or America. Because of Bárány's interest in the subject, Politzer granted him the privilege of using the entire material of the clinic although nominally he was assistant to Alexander in the men's ward.

For critics he did not want. He was surrounded by Politzer, Alexander, Neumann and Ruttin, besides Kreidl and Kubo, the physiologists, each of whom played no mean part in the development of the subject.

For several years before he published his now famous work, he had conducted many experiments on normal individuals and those suffering from various pathologic conditions of the inner ear and eighth nerve.

His attempts were directed principally toward ascertaining the normal reactions from turning and douching the ear with cold and hot water, and how these reactions differed from the normal

in disease. After numerous experiments he was able to give to the world quite definite information on the subject of the physiology and pathology of the semicircular canals from the clinical aspect. Besides, he developed an excellent, though not perfect technic.

Concerning the galvanic test, Bárány makes but slight reference; however, he accepts the experiment of Neumann on the exposed VIII nerve following the labyrinth operation, whereby the nerve reacted to the electrode, but he does not mention the polarity used. He makes brief mention of the work of Volta, who first produced vertigo by galvanism, and of Hitzig, who discovered galvanic nystagmus in 1870; further than this he claimed that the galvanic test up to the time of the publication of his monograph in 1907 was of no practical value.

While Bárány was busy developing his subject, it was the writer's good fortune to be working in the same clinic in the capacity of Zimmerarzt for a year or more under the same chief, Docent Alexander, since made a professor.

The good fortune to have been on the ground floor during this epoch-making period when the subject of the physiology and pathology of the labyrinth was being worked out has served the writer at least three good purposes:

- (1) It taught him careful technic, as practiced by Alexander, Bárány and others of the Politzer clinic.
- (2) A familiarity with the subject from first-hand sources and from its very inception.
- (3) It stimulated him to make further investigations on the subject independently.

The writer has witnessed some work in public clinics and before scientific bodies, so lacking in technic, as to lead to confusing findings, which in turn prompts the examiner to propound still more confusing hypotheses with utter disregard of recognized facts in anatomy and physiology. In the meantime those who have been accustomed to lean on authority lose a great deal of valuable time, and perhaps some lives, before the error is eventually rectified. If but a fractional part of the effort is put into the care of the technic that is put into attempts at explaining away irregular findings, the result of faulty technic, the more rapid will be the progress of scientific medicine. Of the many errors in technic the writer has observed, but few will be cited. Those who have read and observed will recall how Neumann attempted to explain the action of heat and cold on

the inner ear on the principle that heat stimulates while cold inhibits physiologic functions. Had he tried the reaction with the head in different positions before venturing his opinions, as Bárány had done, he would have come to the same conclusion.

Bárány in his excellent work claims that normal individuals react to turning (after ten complete turns) with an after-turning nystagmus of from zero to one hundred and twenty seconds. Now this is positively not so, for in no case has any one else witnessed such tremendous variations in normal individuals. Furthermore, the writer has examined thus far a considerably greater number of normals than Bárány had up to the time of his first publication. On the contrary, the writer's findings have been quite regular. Why did Bárány fall into this error? Because of no other reason than that of faulty technic. He attempted to study the reaction while the patient was permitted to glance to the side, thereby bringing into play a tension on the extra ocular muscles, an error which the writer has pointed out on more than one occasion. For the same reason the Bárány fixation apparatus should be condemned as an instrument that will lead one into error.

Bárány refers to an after-after nystagmus following an after-nystagmus in certain cases. This finding no one else has obtained, including the writer. Yet upon this single observation the result of faulty technic, Bárány and others have busied themselves for several years, formulating theories in their attempts at explaining it.

Any one whose statements are looked upon as authoritative should (a) exercise extreme care in conducting examinations, (b) in the event of an irregular finding in any case, he should prove and reprove it with the object of determining whether the irregularity is not due to some error of technic or to some extraneous influence that may at first have been overlooked, (c) not allow his enthusiasm to displace reason and evolve fanciful theories, and (d) last, but not the least important precaution is, avoid rushing into print with uncorroborated findings and fanciful theories to explain them, because of the danger of misleading the less informed who have been accustomed to look on and accept authorities with implicit faith. Any one may, because of faulty technic, subject a patient now and then to a serious operation with unhappy results when an operation is uncalled for, or may fail to operate when operation is necessary. Such sad happenings are bad enough, but not nearly so bad as when an accepted authority publishes a mass of faulty observations

which are subsequently read, accepted and acted on by the less informed.

SPONTANEOUS NYSTAGMUS

Before making any tests on the semicircular canal it is necessary to note the presence or absence of spontaneous nystagmus. When examining for spontaneous nystagmus it is essential to have the patient look straight ahead at some distant object, after which have him look to the extreme right, then to the extreme left. In the presence of spontaneous nystagmus in any of these positions its character should be noted whether undulatory or rhythmic. If rhythmic, its plane, direction and intensity should be observed and recorded. During the last year, since having heard considerable about the importance of vertical nystagmus as a pathognomonic symptom of cerebellar disease, the writer has been paying more than his usual attention to this form, with the object of corroborating or contradicting the experience of those who have drawn attention to it. Previous to and since then the writer has observed vertical nystagmus upward in a small proportion of normal individuals while looking intently upward.

Spontaneous rhythmic nystagmus to be of semicircular canal origin must be present when the subject looks straight ahead and at a distant object. Any deviation from this direction either increases or inhibits the nystagmus. For instance, if the subject has a spontaneous nystagmus to the left because of an overbalance of impulses in favor of the left semicircular canals due either to a pathologic stimulation from the left side or pathologic inhibition from the right, the nystagmus to the left is increased when looking to the left and diminished when looking to the right. Spontaneous rhythmic nystagmus may rarely be present when looking straight ahead in the case of paresis of one of the extraocular muscles. For instance, in the case of paresis of the left abductor, in rare instances the patient may manifest a horizontal rhythmic nystagmus to the left when looking straight ahead; furthermore, this nystagmus behaves like that produced by an irritative condition in the left external semicircular canal, in that it increases when looking to the left and diminishes when looking to the right.

The writer has observed the frequent occurrence of nystagmus upward when looking upward in those cases showing weakness of one of the elevators of the eyeball. Although normal people show more or less nystagmus when looking in any extreme

direction, the writer has observed that neurasthenics show it to a greater degree.

The spontaneous nystagmus to the left observed in normal individuals when looking to the extreme left may be horizontal or mixed horizontal and rotary. This is a purely physiologic manifestation. The greater the angle deviation required to produce the nystagmus, the greater the tendency toward the rotary direction. The reason for this is more apparent to the eye specialist than to the man who is not familiar with the behavior of the eye.

In making the examination for the presence of spontaneous nystagmus, its character should be noted. Any one should be able to distinguish the difference between the so-called undulatory and the rhythmic forms. As a rule, undulatory nystagmus is considerably more intensive than the rhythmic. By intensive is meant the rapidity and length of the excursions. The most intensive rhythmic nystagmus rarely exceeds the milder cases of undulatory nystagmus. Undulatory nystagmus of ocular origin becomes rhythmic when looking to either side; rhythmic to the left when looking to the left, rhythmic to the right when looking to the right. Occasionally it is rhythmic when looking straight ahead and only becomes undulatory when looking slightly to one or the other side; so that, it becomes somewhat difficult at times to differentiate which type it is unless observed carefully over a considerable period and at different angles of fixation. However, the history of the case including its duration, an examination of the eyes together with the vision and the length and rapidity of the excursion, will aid one in determining its character. While examining the eyes for nystagmus it is important that no handicaps be permitted to interfere with the observations. No one can determine accurately its presence or absence, its character, duration and other important details 3 or 4 feet from the patient. It is essential

- (a) to have good illumination without causing any discomfort to the patient;
- (b) to be near enough to recognize the slightest movement;
- (c) to have the lids of one or both eyes so elevated by the thumb that you can observe the eyeball at the equator, which shows the maximum amount of movement;
- (d) not to have the eyeball uncovered too long at a time, as it becomes dry and painful to the patient, when he is liable to wince or make voluntary movements of the eye, hindering the reaction and interfering with your observation;

- (e) to watch the eyeballs a sufficient length of time to permit you to note the slightest degree of movement; for occasionally the movements are repeated but once in five or six seconds or even longer;
- (f) to study nystagmus with the eyes directed in different positions, i. e. straight ahead, to the extreme right, to extreme left, upward and downward;
- (g) to study all cases with apparently irregular findings a second or third time and have them confirmed by a second equally careful observer, if one is at hand, and
- (h) not to search the brain first to explain these apparently irregular findings but look to some fault in the eyes or to some fault in your technic.

If the patient is resistant or disobeys your directions, wait until he is quieted sufficiently and in a humor to cooperate with you. After the spontaneous nystagmus has been thus studied and recorded, the patient is ready for testing. If his condition is fit for making all the tests, proceed; if not, make a few tests only, allowing him sufficient time between to recover from the strain before proceeding to the next. The tests are far from being pleasant to the patient and he has a right to be considered in the matter. Our object should be to secure as much data as possible for the purpose of making a diagnosis but with the least discomfort to the patient. This has led the writer to adopt the galvanic method in preference to the turning and caloric in those cases where the tolerance of the patient is limited.

Equilibrium and spontaneous past pointing tests should be made before subjecting the patient to the turning, caloric or galvanic tests; otherwise, the patient may be so upset by the latter, that the findings of the former may be somewhat exaggerated. In making equilibrium tests, the first to be thought of is the Romberg, that is, observation of the patient for swaying while he stands erect with feet together with the heels and toes touching. The patient should be directed to stand in this manner some length of time with the eyes closed while the examiner watches him for swaying. Many normal individuals will sway slightly after prolonged standing. The only way to determine pathologic swaying is to observe a sufficient number of normals before passing judgment on pathologic cases. The question of a positive or a negative Romberg can be answered correctly only by one who has had considerable experience. It is not uncommon for one observer to pronounce the Romberg positive in a certain case while another of equal experience feels just as sure that it is negative. Too much is left to the opinion of the observer (personal equation) to allow the writer to accept this test seri-

ously in all cases; therefore, the necessity of making others. In ear cases especially, does the writer feel that it is well to make other modified equilibrium tests including that of standing with the feet together but with the head in various positions; for instance, with head erect, and face forward; with the head erect and the face turned to the right; with the head erect and the face to the left; with the head inclined laterally to the right, that is, with the right cheek to the right shoulder and face directed forward; with the head inclined laterally to the left, that is, left cheek to the left shoulder and with the face directed forward; the head inclined forward and face directed to the floor; the head inclined forward and face directed to the right, and with the head inclined forward and the face directed to the left. These tests have less to do with testing the functions of the semicircular canals than of the vestibular sacs which are especially important as tests for aviators. The Romberg, and all modifications of the Romberg, are static tests pure and simple and would be negative in circumscribed lesions of the semicircular canals and positive in diffuse lesion of the labyrinth. Bárány has made tests similar to these modified Romberg tests after irritation of the canals, but not as a routine measure in suspected cases of labyrinthine affection, as here suggested.

So far as spontaneous past pointing is concerned, the same precautions should be followed. In making them the patient should be tested not only in all directions of movement but also with the head in different positions. Alexander laid stress on the fact and the writer seconded it in his labyrinthine papers long before Bárány published his results on past pointing, that the equilibrium disturbances of cerebellar origin unlike those from pure labyrinthine irritation were constant and independent of changes in position of the head. Bárány carried the experiment further and evolved an excellent series of tests to aid us in cerebellar localization. The writer accepts Bárány's localization tests but with certain limitations. My reasons for so doing are based on some unsubstantiated findings. For instance, it was the writer's good fortune to have recently operated a large subdural cyst of the right frontal lobe, in which case there was pronounced (8 inches or more) bilateral past pointing inward. After the operation the spontaneous past pointing ceased. This experience is further evidence of the recognized fact that symptoms may be produced by pressure on distant parts, first brought

to our attention by MacEwen of Glasgow, and its importance should not be lost sight of in determining, from the symptoms and signs, the location of intracranial lesions (abscesses and tumors).

Every examiner has his own particular manner of studying cases for equilibrium disturbances. Most of them try at least the gait forward and backward with open and closed eyes. Some include hopping forward and backward with open and closed eyes and a few use some form of goniometer. The writer is quite interested in the goniometer (Alexander model) and at one time made extensive tests and published them in the *Archives für Ohrenheilkunde*, 1909, Vol. 7, P. 167. The one thing that impressed the writer at the time and since is that the patient who suffers from an actual disturbance in equilibrium when he falls does so suddenly like something inanimate, like a log, so to speak. He falls so suddenly and unaware, that he has not time to catch himself. The examiner must be on guard, lest the patient is hurt by the fall. Furthermore, on repeating the test many times he invariably falls at about the same angle of elevation. Deaf-mutes from meningitis with complete loss of function of the vestibular apparatus on both sides may walk either broad or narrow. The broad gaited children seem to be steadier than the narrow gaited ones. The width of the gait appears to be less a factor of the disease per se than a manifestation of the child's intelligence. The more intelligent children learn to walk broad, as the sailor or railroader does in his attempts to be on guard against falling. The younger children or those recently deprived of the equilibrium function, walk relatively narrow and more uncertain than the older ones. Furthermore, we may note an individual, who has recently lost his equilibrium function with a narrow, uncertain gait later changing to a broad and more certain one, as he learns of the advantages to be gained thereby. In making tests for gaits one must bear in mind that children who are knock-kneed carry their feet farther apart than normal-legged children.

In testing for equilibrium the deep reflexes should also be studied, for it sometimes happens that the deep muscular sense is lost, for instance, in Friedrich's disease, which would modify to a degree the findings. Allowances should accordingly be made in testing the equilibrium disturbances. It occasionally happens that the patient's gait is even better with the eyes closed than with them open. In such cases the disturbance of equilibrium points to causes located in the eyes or the behavior of the eye

muscles. This subject has been covered rather extensively in former papers by the writer. Any one who is attempting to do work on the labyrinth should be quite familiar with the eyes and the action of the eye muscles; otherwise, he is seriously handicapped, for we find rhythmic nystagmus in certain eye muscle conditions. If one is unable to exclude the eyes as a factor, he can be misled into believing that the same irregular finding of ocular origin is due to a more remote cause in the brain.

TURNING TESTS

Turning tests are more often made with the head erect than in any other position. In order to study the horizontal after-turning nystagmus, ten complete turns equivalent to 3,600 degrees has been generally preferred by examiners. The duration of the reaction to the two sides is compared to the normal and to each other, and from the findings the examiner forms some idea as to the condition existing within the inner ears.

Concerning the position of the head during turning, some assume that since the external semicircular canals are not in the horizontal plane but inclined backward and downward about 30 degrees, it is necessary in order to bring the canals in a horizontal plane and thereby obtain the maximum amount of endolymph motion in these canals, that the patient's head should be inclined forward 30 degrees. The experience of the writer shows that the nystagmus after turning with the head inclined forward 30 degrees is not as purely horizontal as when the patient is turned with the head erect. On the other hand, the writer finds no fault with those who prefer to make the tests with the head inclined forward 30 degrees, but for himself he finds the results obtained by turning the patient with the head erect more satisfactory than with the head inclined slightly forward.

Concerning the number of turns to be tried, Bárány's contention has been that an individual will show a longer duration of after-turning nystagmus after ten turns than after a greater or less number of turns. To this the writer takes serious objection. As stated previously, the greater the number of turns, the longer the after-nystagmus lasts. For instance, the average duration of nystagmus in normal individuals after ten turns is twenty-four seconds. Furthermore, individual differences vary less after twenty turns than after fifteen, and less after fifteen than after ten turns. In short, considerably more reliable results are

obtained after twenty than after ten turns. This statement is based on the results of numerous experiments on both normal and pathologic cases. Variations of slight amount, that is to say, six or seven seconds from the normal or variations of a like amount on the two sides after ten turns, should be allowed for errors in technic, since there is less variation to be found on the two sides after fifteen or twenty turns than after ten turns. I would suggest to those obtaining marked discrepancies in the duration of after-nystagmus to the two sides after ten turns that they repeat the tests making use of a greater number of turns, preferably twenty, when they will frequently be gratified with more accurate and consistent findings. A variation greater than seven seconds after ten turns is suggestive of something pathologic in the labyrinth or nerve but not necessarily an indication of destruction. A decrease in the duration of the after-nystagmus to one-third the normal on one side and to two-thirds the normal on the opposite side is more than suggestive of a destructive condition within the semicircular canal apparatus of the side corresponding to the greater diminution provided the other side is known to be normal.

Horizontal after-nystagmus is the one most often studied by the average examiner and for ordinary purposes it is quite sufficient so long as the findings are normal; but otherwise not. For instance, in the case of a circumscribed destructive lesion in the external semicircular canal of one side, the duration of the horizontal after-nystagmus to the two sides, and especially to the affected side, may be sufficiently reduced as to indicate a destructive process in the inner ear. How are we to determine by the turning test alone whether the lesion is limited to the external canal or involves the entire labyrinth? To determine which of these two conditions is present, it is necessary to turn the patient with head in other positions, the preferred position being with the head inclined forward 90 degrees or more. In this position there is produced a rotary after-turning nystagmus in the direction opposite to that which the patient has been turned. This rotary nystagmus is due in normal individuals to the irritation of four canals, that is, the two verticals of the two sides. Now, if the case is one in which there is a localized lesion in the external semicircular canals, the rotary nystagmus to the two sides will balance quite well, whereas if the lesion is a diffuse one, the rotary after-nystagmus to the two sides will be characteristic of that found in a lesion of this type, that is, a pronounced reduction to the affected side and moderate reduc-

tion to the unaffected side. If it is necessary to turn a patient, at all, in order to determine the function of the inner ear, a twenty turn test will produce only a slightly longer duration of the nystagmus and discomfort than ten turns, and at the same time will give more accurate results than a ten turn test. The patient is thereby relieved of the necessity of having to repeat the test where the results might have been doubtful after ten turns. In other words, the average patient would prefer being rotated twenty turns to one side followed by twenty turns to the opposite, notwithstanding suffering a few seconds longer with vertigo, than to be turned ten times to each side and at the end of the experiment be obliged to have it all repeated in order to confirm the first findings.

It is rarely necessary to turn the patient in any other than these two positions, that is, head erect and head inclined forward 90 degrees. However, to do so makes interesting experiments but at the cost of the patient's comfort. On the other hand, it should be practiced in any critical case when necessity calls for it or when important information cannot be had by some easier method. It should be remembered that after turning the direction, the intensity and the duration of the nystagmus is not materially interfered with by a moderate movement of the head. There is no reason, therefore, why the rotary after-nystagmus should not be studied and timed with the patient's head in the upright position or inclined slightly backward with good illumination, rather than in the primary position with the examiner a yard or more from the patient with poor illumination, as witnessed by the writer in some of the clinics. Such methods comprise poor technic, and are responsible for the presentation of this paper.

CALORIC TEST

The caloric test is truly a unilateral test which is a distinct advantage it holds over the turning test. On the other hand, it lacks one advantage of the turning test in that it is not a quantitative one.

By the caloric test we are able to settle the problem of whether the semicircular canals are completely destroyed or not, of the side on which the test is made. Further than this it tells us nothing. It can not tell us whether the function is above normal in cases of congestion of the labyrinth nor can it tell us that it is below normal, so long as there is some remnant of function present.

Its especial value is in aiding us to determine whether the labyrinth is completely destroyed or not, for instance, in cases of suspected suppuration of the labyrinth.

It is conceded by every one that, when a sufficient amount of cold water is applied to the external canal in normal individuals, a rotary nystagmus to the opposite side follows so long as the head is held in the upright position. Furthermore, that hot water produces the opposite effect, that is, rotary nystagmus to the same side. These reactions are quite generally understood.

Now there are a few things that should be borne in mind:

(a) That in the presence of large polypi, cholesteatomata, foreign bodies, abscesses and other obstructions in the canal, the reaction is delayed and occasionally negative. In other cases of moderate obstructions the reaction is merely delayed. In either case one should not be too quick to blame the lack of reaction to a faulty labyrinth rather than place it where it properly belongs—in the external canal. The writer has witnessed quite a number of cases in which there was negative reaction which became positive after the removal of obstructions from the external auditory canal.

(b) That cold water is tolerated better by the patient than hot, and therefore is better adapted for making the caloric test.

(c) That in those cases in which the test is of greatest value (labyrinth suppuration), the patient has already a spontaneous nystagmus to the side away from the lesion. Since suppuration of the labyrinth, let us say of the right side, produces a spontaneous nystagmus to the left, cold water applied to the right ear, in the presence of a reactive labyrinth, can only increase the existing nystagmus so long as the head is held erect. On the other hand, if the inner ear is destroyed, there is no change in results. The observer is left to judge whether the nystagmus is increased or diminished, and in many cases this is no easy matter. The writer has witnessed on more than one occasion two or three experts in the Politzer clinic, Bárány among them, attempting to decide whether an existing spontaneous rotary nystagmus to the opposite side was increased or not from the application of cold water, eventually resulting in a debate and further experiments on the patient. In recent cases of labyrinth suppuration when the rotary nystagmus is quite pronounced, no amount of cold water could possibly increase the existing nystagmus.

The natural question arising in one's mind, under such circumstances, is: Of what value then is the caloric reaction conducted after the current fashion? Would it not be a more satisfactory plan to attempt the caloric test after a manner that eliminates all guess work? Such a method has been tried successfully and fully described by the writer seven years ago.

For testing the right ear, incline the patient's head to the left shoulder so that the left ear rests on the left shoulder and the face is directed forward. In this position the effect of cold in the right ear is to produce a horizontal nystagmus to the right if the inner ear is reactive. A complete change in the direction of the nystagmus can leave no doubt in the observer's mind. It is not necessary to syringe the ear with the patient's head inclined laterally to the shoulder to obtain this reaction, for it is characteristic of the caloric test that its plane and direction is changed by changing the position of the head. The caloric test may be tried in the usual manner, that is, with the head erect, and if after a reasonable trial with cold water, there is any doubt as to the presence of a reaction, all that is necessary is to place the head in the position suggested in the foregoing when there follows a horizontal nystagmus to the same side of sufficient intensity as to leave no room for doubt in the observer's mind.

The writer has witnessed experiments conducted by men of national reputation who attempted caloric tests with the head erect and then subsequently with the head inclined backward. Now with the head backward the spontaneous rotary nystagmus to the opposite side is changed to that of horizontal. The plane of movement changes, but not its direction, which again may lead to some confusion. By changing the position of the head after the manner suggested by the writer, both the direction and plane of the nystagmus are changed, which leaves the least possible chance for error.

FISTULA TEST

Concerning the technic of the fistula test, there is not a great deal to be said and no criticism to make of the manner in which it is generally conducted. It is a unilateral test and the position in which the head is held has no influence on the reaction.

The reaction is present to a mild degree in many normal individuals; however, to a stronger degree in certain pathologic cases. On the other hand, the reaction may be negative in the presence of a fistula. But in spite of these apparent objections, the reaction when present and marked is of positive value. The typically positive reaction is produced by compressing the air in the external canal and middle ear when it manifests itself in the form of a nystagmus to one or the other side, depending on the location of the fistula. A somewhat less intensive nystagmus is produced by suction but to the side opposite to that produced by compression. For instance, a fistula of the osseous external

semicircular canal with an intact membranous canal will react to compression to produce a pronounced horizontal nystagmus to the same side, while suction will produce a horizontal nystagmus somewhat less intensive to the opposite side. A fistula in the region of the vestibule will, on compression of air, produce a pronounced rotary nystagmus to the opposite side while suction will produce a less pronounced rotary nystagmus to the same side.

Concerning a fistula of the external semicircular canal, the reactions are quite definitely settled. Concerning the other parts of the labyrinth, there is still some doubt owing to the scarcity of accurately studied material by laboratory methods. There is no scarcity of recorded cases from the clinically studied standpoint. It is well to bear in mind that a fistula of the osseous labyrinth may not yield the characteristic findings of fistula, because of a coincident destruction of the membranous labyrinth. Besides, in the cases of an overly patulous eustachian tube the air may escape so effectually by this route as to hinder any attempts at piling up pressure in the middle ear sufficient to produce the symptom in spite of otherwise favorable factors. In some cases the symptom may be produced by simple pressure of the tip of the observer's finger in the external canal of the patient. In such a case there is a strong probability that in addition to a fistula there are polypi, granulations or cholesteatomata present and that these are pushed directly into the fistula and thereby produce the symptoms.

Concerning the technic of the galvanic test, since the writer has so recently covered the subject and rather fully, he will postpone any further discussion of it at this time.

In closing, the writer wishes to emphasize one of his previously expressed thoughts on the subject, namely, that the galvanic test properly conducted is the most satisfactory of them all for it combines the favorable features of both the turning and the caloric tests.

DISCUSSION

DR. JOSEPH E. WILLETTS, Pittsburg: I was very much surprised on receipt of the program to see that I was down for the opening discussion of this paper of Dr. Mackenzie's. Dr. Mackenzie is a pioneer in the study and examination of the labyrinth, and as the subject is especially pertinent at this time, I feel that some member better qualified than I should have been selected. When one considers the complicated apparatus to be examined, it is easy to realize that the possession of a revolving chair does not make one an authority on the subject. There are many things one must keep in mind in considering results. The gross anatomy and physiology of the labyrinth to me seems to be the most important. The past pointing is a result of the vertigo produced by an

endolymph movement, and not by the movement of the endolymph itself. While this seems to be a rather fine point of distinction, nevertheless, it shows that past pointing is not a reaction of endolymph movement, but that it is dependent on unusual phenomena produced by unusual procedures. If these unusual procedures be repeated sufficiently often for the cerebellar coordination centers to become accustomed to the phenomena produced, the past pointing and nystagmus diminishes, and if they be continued for any length of time, they do not appear at all. Two instances I might here cite:

In Paris a number of years ago, I, like all good Americans, visited the Moulin Rouge. During the evening a young man and girl started to dance and the spectators gathered around them. The dancers placed their arms around each other's waist and spun around on their toes in a most amazing fashion for fully five minutes. I do not think they varied a foot in floor space. I spoke to one of them (which one does not matter) as soon as they stopped. Her eyes showed absolutely no sign of nystagmus. The incident has been recalled because of the similarity of movement to that of the revolving chair as used in the test described. The Dervish dancers revolve for much longer periods with very little confusion of coordinating centers. We can see that repetition modifies the result, and it seems to me that Dr. Mackenzie's suggestion to increase the length of time and number of turns on first examination is a logical one, and from the same deductions, for the sake of standardizing and expressing results, the pointing should be confined to but three trials at regularly stated intervals by the watch, after each turning.

The technic of any operation is the result of logical deductions from the study of the conditions and the anatomy of the part to be operated on. The technic of the labyrinthine test must therefore, it would seem to me, conform to the anatomy or the position of the semicircular canals; otherwise the signs produced by the test must be mixed and worthless for record. Logically the position of the patient's head, under examination, during rotation, should conform to the position or plane of the semicircular canals under examination. The Germans, who originated this test some five or six years ago, I am told, failed in this respect, and their results, observations and records were faulty. The external or so-called horizontal canal inclines downward and backward. To bring this canal in the horizontal plane parallel with the floor, the head must be inclined forward 30 degrees. Rotation to the right in this position brings a clean, unmixed nystagmus to the right. Dr. Mackenzie's statement that you get the same result with the head in the vertical position, is to me most perplexing, for in this position none of the canals are in the horizontal plane, yet you have a horizontal nystagmus. If the plane of the canal under examination needs no consideration, what is there about the technic as a whole to tie to? The posterior semicircular canal and the superior semicircular canal, the so-called vertical semicircular canals are not vertical. But these two canals communicate with each other and the vestibule at an angle through a single orifice on its posterior wall. Now it is well known that two semi-solid currents whose point of impact is at an angle, the impulse from the point of impact becomes median. The impulse then during rotation, as conveyed, is midway between the two vertical canals and is in effect and might be considered as a single canal midway between the two and in a perfectly vertical meridian. Now what happens when a vessel containing a fluid is forcefully revolved in the vertical meridian? The force and flow of the fluid is toward the periphery. The force as applied is centrifugal. As this is a fixed law, the contents of the vertical canal or canals during rotation

for examination of the horizontal canal must be depleted or emptied of their contents, and out of commission as far as interference is concerned.

During rotation with the head inclined forward or backward to 90 degrees the external or horizontal canal becomes in this position vertical and is out of commission through its subjection to the same influence. The vertical canals, with the head in this position are now brought on to the horizontal plane. But we now have two canals on each side, instead of one canal to consider. These two canals are at an angle to each other, and while they are in the horizontal zone, they are not parallel, one being superior and posterior to the other, but they communicate with each other. In this new position they are out of the centrifugal zone, and the current of endolymph through them is as induced by the direction of rotation. Two arched canals, almost completing a circle, not in the same plane, communicating with each other, must, during rotation, excite a current through both.

I would like to ask Dr. Mackenzie whether the circulation of endolymph in the vertical canal, when in the horizontal position, is conceded to be a circular or lateral current? I really have not had the time to look the matter up. One of these canals is higher than the other. They communicate. Their plane is not relative. So one of them must necessarily be subjected to a greater momentum during rotation, and the current in that canal relatively faster. Slight as this may be, it would excite a tendency to a circular current, as compared with the straight left to right movement of the endolymph in the single compartment of the horizontal canal when examined in the same plane. The nystagmus of the vertical canals is rotary. The nystagmus of the horizontal canal lateral, though both be in the same plane.

DR. OTTO J. STEIN, Chicago: Regarding the past pointing test, I have found it difficult sometimes to correctly measure the time after turning for the past pointing, on account of the difficulty in bringing the patient's finger back into the correct median line of the operator's supposedly fixed finger. I have substituted a dummy or wooden finger on a stand. This is just a suggestion as to how I have been trying to cut out a loss of time and get more accurate results.

DR. H. H. FORBES, New York City: I wonder if the general profession realize the work that Dr. Hayden and Dr. Mackenzie have devoted to this important subject. Dr. Hayden has taken it in an entirely different way, and from the standpoint of an examination for the aviation corps of the United States Army. In New York we have done a little of this work. The young men who have come before us for these examinations have been in the pink of perfection physically. No one has been turned down for a failure in any one of these tests Dr. Hayden has gone over.

My reason for sending in my name is to tell you that we are just starting in this work. The tests given here are all right as far as they go and the men passing may go into the flying corps and make a success, but it has been found that a number who pass these tests do not make good flyers. There is some other physiologic function which we have not discovered. There is a machine which tries to imitate some of the conditions the flyer will be meeting in flying recently devised by a Chicago physician. By means of a cabinet one is able to simulate the actual conditions as regards altitude up to 3 miles. The man is raised by exhausting the air in a cabinet. I believe they can get down 2 or 3 miles very quickly, between thirty-eight and forty seconds and the man can be brought back to normal atmospheric pressure in this time. In a number of these cases when men have come down it is impossible to discover any mechanical defects, but it is being worked out on the line

that maybe it is muscular fatigue and that the atmospheric conditions will excite him in such a way that he lacks muscular tonicity after a certain time in the air. It is a wonderful idea as I saw it used, and might be added in making these tests.

DR. J. H. MCCREADY, Pittsburg: It is surprising sometimes how accidents will demonstrate things. Last week a man, aged 35, went out to dinner with friends, ate heartily and drank wine. He got up from the table and knocked three or four chairs over and every one thought him drunk; after while they were convinced something was seriously wrong. He had a divergent squint. He saw an eye specialist and it was found that he had a rotary nystagmus horizontal. He came to us and we found also a spontaneous vertical nystagmus. We tested him by past pointing, etc., and localized a lesion in the upper part of the pons. This shows how you may put your finger on many lesions by different tests. If turned to the right he would not fall, but if turned to the left he would. We put him to bed and kept him quiet and it cleared up. It must have been a very small hemorrhage.

DR. HAYDEN (closing discussion): Dr. Stein remarks about the wooden stiffer finger, which is certainly an advantage, having a finger which does not deviate. But if we use any sort of wooden finger it must be moved, that is, when the right arm is being examined the finger should be put perfectly in line in front of the body.

It is perfectly true that the European governments do not require these tests, and it is also true that some of the men we have examined and pronounced fit are not fit. But you must remember that these tests have only been made for the past few months. That the series of cases reported here this morning is the largest observed, and it is very essential that all of us become interested in this work and compile as many as we can so the findings may be recorded with all the clinical data obtainable. The United States Government requires men absolutely perfect. They would not require that if we had been in the war as long as France and England. When men become as scarce as that the requirements will lessen and the less fit will be called to take the place of the men who are now supposed to be 100 per cent. efficient.

DR. MACKENZIE, Philadelphia (closing discussion): I agree with Dr. Willets that those individuals who have been accustomed to being turned will show a less degree of nystagmus and past pointing than those individuals who are being turned for the first time. I had occasion to examine a whirling Dervisher for after-nystagmus and past pointing. He was a performer in vaudeville and was accustomed to turning for many minutes at a time. I examined him on several occasions and found that he could turn hundreds of times without becoming dizzy but nevertheless he would manifest the same character of after-nystagmus as a normal individual but much shorter to the left side than to the right. His exhibitions were always right turning. On examination he manifested no spontaneous nystagmus or past pointing nor could any irritation of his semicircular canals cause him to do so, for he had learned to ignore the sensation of vertigo and to correct the tendency to past pointing.

Dr. Stein, I believe, mentioned the importance of taking the past pointing test at an exact time after ceasing the turnings, for only then can we expect to obtain accurate and consistent findings; for instance, the past pointing in any normal individual will be more pronounced during the first few seconds than when taken after a greater length of time. Concerning the question raised by Dr. Willets as to the most favorable position for the head to be placed in during the turning in order to produce a purely horizontal after nystagmus, I wish to say that the location of the semicircular canals in space is not a definite guide as to the char-

acter of the nystagmus, as the position of the head for the nystagmus follows the rule with one exception that the plane of the nystagmus is in the plane of turning and the direction of turning during turning and in the same plane in the opposite direction after turning. The exception to this rule is when the patient's head is thrown backward 45 degrees between the erect and horizontal; that is, midway between the vertical and horizontal position. In this position we note an after turning nystagmus horizontal to the opposite side combined with rotatory to the same side. This peculiar combination was ascertained by Bárány among his earlier experiments. It is generally accepted that when the patient's head is directed forward toward the floor there is produced a rotatory after nystagmus to the opposite side after the patient has been turned. Now, if we are to accept the horizontal canal as normally inclined backward and downward 30 degrees from the horizontal plane and that the vertical canals are at right angles to the horizontal, then it follows that with the head inclined forward 90 degrees, there is not a single canal that is in the exact horizontal position nor at right angles; besides, during the turning the three canals on the two sides are stimulated. Now to figure out which particular canal was responsible for the rotatory nystagmus would lead to considerable confusion, nor is it necessary for practical purposes to know exactly which canal plays the greater part in the production of nystagmus in any plane so long as we remember the rule that the after nystagmus is in the plane of the turning and in the direction opposite to that turning. To irritate by turning a single canal we would need to select a case in which one side is known to have been destroyed and attempt to place the patient's head in such a position as to produce the maximum amount of endolymph motion in one canal only. For instance, let us take a case of complete destruction on the left side with the right side intact; in order to produce in this case endolymph motion in the superior semicircular canal only, the patient would need to be turned with the head inclined forward and somewhat more than 90° (approaching 120°) and with the face directed to the right 45°. To test the posterior semicircular canal, it would be necessary to have the head inclined forward and somewhat more than 90° and with the face directed to the left 45°. In order to produce the maximum amount of endolymph motion in the horizontal canal, it would be necessary to have the head inclined forward 30° or less with an inclination of about 5° to the opposite shoulder. For scientific purposes pure and simple such experiments would be interesting, but for practical purposes they are unnecessary, and such care has never been taken by any examiner up to this time.

Marked discrepancies in the findings of after turning nystagmus generally speak for poor technic, as I have pointed out on previous occasions. It is very essential to have the patient look at some distant object straight in front of him, for a slight deviation of the eyes to one or the other side will have a tendency to lengthen the duration of the nystagmus to the side to which the eyes deviate and diminish it to the side from which the eyes deviate. Furthermore, the more experienced observers are likely to find a slightly longer nystagmus than the less experienced since they have learned to recognize the finer eye movements better.

A NEW DIAGNOSTIC SIGN OF FOREIGN BODY IN THE TRACHEA OR BRONCHI, THE "ASTHMATOID WHEEZE"

CHEVALIER JACKSON, M.D.

PHILADELPHIA

It is needless before a meeting of this character to elaborate on the necessity of developing to the utmost all diagnostic means for use particularly in cases of foreign bodies not opaque to the roentgen ray. It is also unnecessary to do more than allude to the well known fact that pathologic shadows often show the presence of a foreign body that is itself not opaque to the ray. The absence of such shadows, however, we know, are not an indication that a foreign body is not present. Auscultation and percussion are, of course, extremely valuable in many cases, though here also the negative report is much less valuable than if positive. It is a great misfortune that in many cases of foreign body of great density, such as metallic bodies, and showing clearly in the ray, auscultation and percussion have been neglected. The author has endeavored to obtain records as complete as possible in all of his cases for their statistical value regardless of whether the ray was positive or negative. To the well known physical signs he has added a new one, which he has called the "asthmatoïd wheeze." He regrets, however, that his statistics on this sign do not go back as far as his other statistics.

IMPORTANCE OF THE SIGN

The presence or absence of the "asthmatoïd wheeze" has been recorded in sixty-two cases. From this record the author feels that we have a new sign that promises to be of great value in many cases in which the foreign body is not opaque to the ray. He has called this new sign the "asthmatoïd wheeze" because the sound is somewhat similar to the wheezing heard when the ear is placed to the open mouth of an asthmatic patient. The chief difference is that there is more or less association of the sounds of râles with the wheezing in asthma, while in the asthmatoïd wheeze of the foreign-body case the sound is dryer, when typically elicited, though of course it may be associated with more or less secretion, adding more or

less of the sounds of bubbling. In a typical case, however, such as No. Fbdy. 623 reported in this article, the wheeze is much more marked after the coughing out of all secretion than before. This phenomenon is quite likely due to the secretions more or less completely occluding the spaces by which air escaped between the wall of the bronchus and the irregularly-shaped foreign body itself. When these irregular air passages are cleared out, more air passes, and consequently the wheezing sound is louder.

The author has deemed it best to call the attention of his confrères to this sign without waiting longer for accumulation of data in order that its value may be determined by the largest number of observations.

Numerous cases are reported in the literature in which audible sounds in foreign-body cases have been studied as heard with stethoscope or naked ear applied to the chest wall or over the cervical trachea or larynx, but I have been unable to find in the literature any mention of a study of the sounds as emitted from the open mouth. The nearest approach to it is the often mentioned "croupy cough" indicative of a laryngeally lodged foreign body; but even in these cases there is no mention of the study of the respiratory as distinguished from the bechic sounds. It is, however, not in the laryngeal cases that the asthmatoïd wheeze is elicited. It is in the tracheal and bronchial; hence the great importance of the sign. If there is a croupy cough, the larynx must be looked at. This is readily done and the matter of laryngeally lodged foreign body readily decided, positively or negatively; but the indications for bronchoscopy in suspected foreign-body cases are often not so clear, and one feels that the indications in some cases are worthy of more thorough study than in a laryngeal case before a decision to do, or not to do, a bronchoscopy is reached.

CHARACTER

The asthmatoïd wheeze may be defined as a sound heard by placing the ear in front of the patient's mouth during expiration. It resembles the wheezing of the asthmatic patient but is dryer. It is caused by the vibration of the air passing the foreign body in the bronchus. It is elicited by placing the ear in front of the open mouth of the patient while breathing deeply and regularly, in and out. If the wheezing is not noticed the patient is asked to carry respiration to the extreme limit as though endeavoring to expel all of the residual air from the lungs. In a number of instances the wheezing has been apparent only at the termina-

tion of this prolonged, forced inspiration. In many other instances the wheezing was so loud that it was noticed all over the room. In eliciting it, care is needed to distinguish between a wheezing caused by secretion and a wheezing caused by a foreign body. This cannot always be done with certainty. Moreover, more or less secretion is usually present in the neighborhood of the foreign body. Usually the sounds of secretion disappear after coughing and expectoration, while the wheeze of a foreign body remains after the most violent and persistent coughing.

The pitch and loudness of the sound seem to depend on the size, position and shape of the foreign body, as on these characters depend the size and shape of the spaces through which the expired air passes the foreign body. In one case the wheeze terminated in sharp slapping sound due to the impact of the suddenly arrested foreign body which had been dislodged.

LOCALIZATION

The asthmatoïd wheeze is of no localizing value. Being a sign heard at the open mouth, not with the ear at the chest wall externally, it is impossible to determine out of which bronchus the wheeze comes. In one case a foreign body in the trachea gave a slightly different note, very much flatter in tone as compared to the note as heard in cases of bronchially lodged foreign bodies. Whether or not further observations will establish this to be the case, or if so whether or not it will be possible to determine by the absolute pitch, whether the foreign body is located in trachea or in bronchi, it is impossible to say at present. Observation by those having well trained ears will be very valuable here.

PHYSICAL CHARACTER OF FOREIGN BODIES PRODUCING THE ASTHMATOÏD WHEEZE

Smooth rounded foreign bodies that cork a bronchus tightly have not, in any instance, produced the asthmatoïd wheeze, properly so called. Many of the cases associated with considerable amounts of secretion in the trachea and bronchi have caused a moist wheeze, but this sound is of less diagnostic value. The sound was elicited clearly and sharply in cases of foreign body of larger diameter than a pin and especially in those of angular shape such as bones, nut-shells, grains of corn, and the like.

MECHANISM OF THE PRODUCTION OF THE ASTHMATOID WHEEZE

This is, of course, conjecture. It seems quite probable that the sound is produced by the air passing the foreign body; in other words, by passing through a very much diminished area of cross-section, and passing over, or impinging on, rough, sharp surfaces, instead of passing, as it normally does, through the round, relatively smooth mucosa-covered air passages.

REPORT OF CASES

To report in detail all of the cases in which the asthmatoïd wheeze was searched for, would needlessly prolong this paper. The test was made in sixty-two cases. The wheeze was present in forty-one cases of foreign body, absent in twenty-one cases in which the foreign body was afterwards discovered or previously known to be present. The following cases are typical of the classes in which the wheezing was present and absent respectively:

CASE 1 (No. Fbdy. 624).—*Pebble removed by oral bronchoscopy from right bronchus. Asthmatoïd wheeze not present.*—*History.*—J. W., a girl, aged 4 years, was referred by Dr. O. W. Saunders, Camden, N. J., for the bronchoscopic removal of a pebble aspirated two days previously. The child had been playing with pebbles, a number of which she had in her mouth. Following the aspiration there was dyspnea, coughing, vomiting and blood-streaked sputum at irregular periods. Dr. Edwin E. Graham reported diminished expansion and harsh breath sounds from the upper right anterior portion of the chest.

Roentgenographic Report.—The roentgenogram made by Drs. Manges and Bowen was reported on as follows: "In the case of J. W. there is a remarkable condition in that the foreign body is surely in the right bronchus with the right lung showing completely aerated, whereas the left lung casts a considerably more dense than normal shadow as if there had been some inflammatory process in the left side. It occurs to me that probably the foreign body was at first lodged in the left bronchus and remained there until a comparatively short time before the roentgen-ray examination was made. After the foreign body was removed there seemed to be less difference in density between the two sides but still a shade of difference. This difference in density of the two sides might, so far as the plate is concerned, be due to overdilatation of the right lung with air; in other words, a temporary emphysema. If it were possible that this pebble could have acted as a ball valve admitting air but not letting it out the results would accord with the appearance on the plate."

Examination.—On testing for the asthmatoïd wheeze by put-

ting my ear to the patient's open mouth I was unable to detect any abnormal sound.

Bronchoscopy without anesthesia, general or local, revealed the pebble tightly wedged in the right main bronchus above the upper lobe bronchus, from which it was disimpacted and removed in three minutes and fifteen seconds.

Comment.—Recovery was prompt and complete. The absence of the wheeze in this case was doubtless due to the tight, cork-like fitting of the pebble in the bronchus preventing escape of any air past the foreign body.

CASE 2 (No. Fbdy. 623).—Bone removed by oral bronchoscopy from right bronchus. Asthmatoïd wheeze diagnostic.—History.—A man, aged 39, was referred by Dr. Schlindwein, Erie, Pa., with a history that while eating beef soup he had gagged on finding a bone in his mouth which he was unable to extrude. A deep inspiration aspirated the bone. The immediate symptoms were: laryngeal pain, severe coughing, blood-spitting, wheezing, substernal pain and labored respiration. An unsuccessful bronchoscopy had been done before the case reached Dr. Schlindwein, being followed by a temperature elevation to 104 F., severe coughing, foul sputum, increased spitting of blood and dyspnea, with total aphonia. An excellent roentgenogram (considering the enormous size of the patient's chest) failed to show any evidence of foreign body. The larynx was not stenosed, though lacerated.

Examination.—Physical examination of the chest by Dr. W. H. Spencer was reported as follows: "Thorax rounded. Thick walls. Expansion poor, seems equal. Percussion seems clear—slightly impaired generally because of thick chest walls. Breath sounds distinct. Expiration slightly prolonged over upper right. Many sonorous and sibilant râles over whole right chest. Most plainly heard below angle of right scapula, where there is the suggestion of a friction sound. Few softer bronchial râles heard in left axilla and left upper chest posteriorly. Respirations wheezy and asthmatic."

These findings were confirmed by Dr. E. Quin Thornton, who gave the following opinion: "There is evidently an inflammation of the bronchial system of the right side with slight involvement of the left side." This opinion was confirmed absolutely at bronchoscopy.

Treatment and Results.—In view of the negative roentgenogram and traumatic laryngitis the question arose as to whether or not bronchoscopy should be done. The decision to do it was based solely on the asthmatoïd wheeze, which was so marked that it could be heard anywhere in the room when the patient had his mouth open. The wheezing sound had not the character of râles, evidently being produced by the foreign body itself and not by the attendant secretions. The wheezing was greater after expectoration than before.

The bone was removed bronchoscopically under local anes-

thesia in fifty-eight seconds exclusive of the time required for applying the cocain. After removal of the foreign body Dr. Spencer reported: "Wheezing respiration and the bronchial râles have disappeared. Respiratory sounds over the lower and middle right lobes somewhat harsher with respiration more prolonged than on the left side. Patient has no dyspnea and is very comfortable." The patient was discharged the next day and made a complete and uneventful recovery.

Comment.—This may be regarded as a typical and as the leading case, though not chronologically first, in the establishment of the asthmatoïd wheeze as an important diagnostic sign of foreign body in the bronchi. The roentgenogram was negative; the asthmatoïd wheeze was very loud; it was louder after effective expectoration; bronchoscopy was done on the strength of asthmatoïd wheeze positive; the wheeze disappeared totally immediately after bronchoscopic removal of the foreign body.



Fig. 1.—Part of the glass eye of a "teddy-bear" that, as a foreign body in the bronchus of a child of five years, produced a typical "asthmatoïd wheeze" (Case No. Fbdy. 583).

CASE 3 (No. Fbdy. 583).—Glass eye of "teddy bear" removed from right stem bronchus by oral bronchoscopy. Asthmatoïd wheeze present.—History.—A girl, aged 2 years, aspirated half of the glass eye from a "teddy bear." The immediate symptoms were dyspnea and choking; later symptoms were coughing and wheezing.

Examination.—Physical examination of the chest by Dr. Elterich showed that a very much diminished amount of air was entering the right lower and middle lobes. On placing my ear to the open mouth of the patient the asthmatoïd wheeze was very plainly perceptible on expiration. A roentgenogram had been taken but it failed to show the foreign body. The patient was then bronchoscoped, revealing the presence of the foreign body in the right bronchus, which, however, the operator was unable to remove. He referred the case to Dr. J. W. Cheney, Wichita, Kan., who referred the case to the author. A roentgenogram by Drs. Johnston and Grier showed, not only the glass eye, but in addition the pupil of the eye was differentiated from the iris clearly in the roentgenogram. Bronchoscopy without anesthesia, general or local, promptly revealed the presence of the foreign body presenting in conoidal form.

Treatment.—It required some manipulation properly to seize the foreign body for removal. The whole procedure from the time of the introduction of the bronchoscope required five

minutes and fifteen seconds. The wheeze disappeared totally after bronchoscopic removal of the foreign body. (Fig. 1.) The child left for her home the next day, and the recovery was complete and uneventful.

Comment.—It is not always easy to get a child of 2 years to cooperate in eliciting the asthmatoïd wheeze; but patience accomplished it in this case. It was plainly noticeable also when the child slept.

CASE 4 (No. Fbdy. 579).—*Grain of corn removed from right bronchus by oral bronchoscopy. Asthmatoïd wheeze present.*—*History.*—A girl, aged 5 years, was referred to me by Dr. J. W. Mayhew, Lincoln, Neb., for the removal of a grain of corn aspirated while playing. The immediate symptoms were violent coughing and dyspnea for about fifteen minutes. Later symptoms were occasional dyspnea and coughing.

Examination.—Physical examination by Dr. F. T. Billings revealed very much diminished expansion of the right lung during inspiration with dullness on percussion all over the right chest. On placing my ear to the child's open mouth the asthmatoïd wheeze was plainly heard. It was much clearer after expectoration than before. Roentgen-ray examination was negative.

Treatment.—On passing the bronchoscope without anesthesia, general or local, the grain of corn was seen impacted in the right bronchus from which it was readily removed. The time required for the introduction of the bronchoscope and removal of the foreign body was two minutes and fifty-four seconds. After bronchoscopy the asthmatoïd wheeze had altogether disappeared and all the abnormal physical signs were absent except some moist râles. The child left for home the next day and made a prompt and uneventful recovery.

Comment.—In cases such as this the asthmatoïd wheeze is of very great importance because of the negative result of the roentgenogram. However, Dr. Mayhew had found the physical signs so strongly indicative of foreign body that he had advised the child coming over 1,000 miles for the removal. It is not every diagnostician who feels so positive as to his diagnosis of foreign body in the lung.

CASE 5 (No. Fbdy. 560).—*Wooden whistle removed from left bronchus by oral bronchoscopy. Asthmatoïd wheeze present.*—*History.*—A boy, aged 8 years, was referred by Dr. Rush, Portage, Pa., with a history of having aspirated part of a wooden whistle.

Examination.—A roentgenogram was entirely negative. Dr. H. T. Price found râles in the left chest with very harsh breath sounds. His opinion was that there was a foreign body partially obstructing the entrance of air in the left lung. On placing my ear to the patient's open mouth a very distinct asthmatoïd wheeze was plainly heard on expiration. It was very much increased at the end of expiration when the patient forcibly expelled all that was possible of the residual air.

Treatment.—On passing the bronchoscope I found the wooden whistle (Fig. 2) tightly impacted in the left bronchus. I could see plainly into the tubular end though momentarily obstructed by a small fragment of rubber still adhering to the whistle. An expanding forceps was inserted into the lumen of the whistle which was then readily withdrawn. The child was discharged the next day and recovery was prompt and uneventful.



Fig. 2.—Wooden cylinder that, as a foreign body in the bronchus of a boy aged 8 years, produced an "asthmatoïd wheeze" (Case No. Fbdy. 560).

Comment.—The asthmatoïd wheeze in this case was not produced by any mechanism similar to that of a whistle. The foreign body consisted simply of a piece of wood 3 cm. long by 8 mm. in greatest diameter, with a hole throughout the long axis. As a toy the sound is produced by the vibration of a rubber accessory. The asthmatoïd wheeze had a very slight cooing sound possibly due to the smooth round hole through which the air was passing. The asthmatoïd wheeze in this case was characteristic only at the end of prolonged and forced expiration.

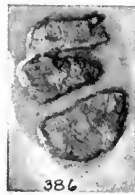


Fig. 3.—Three pieces of fish bone that, as foreign bodies in the bronchi of a woman aged 25 years, produced a typical "asthmatoïd wheeze" (Case No. Fbdy. 386).

CASE 6 (No. Fbdy. 386).—*Fish bone removed from left inferior lobe bronchus by oral bronchoscopy. Asthmatoïd wheeze present.*—*History.*—A woman, aged 25, was admitted to the Montifiore Hospital, Pittsburgh, with a wheezing respiration but no cyanosis. She stated that she had choked on a piece of fish

bone, the accident being followed by a severe attack of coughing. There were no other symptoms until about two weeks later when she began to expectorate pus and the temperature began to go up a little every day, the maximum being about 99.4 F.

Examination.—Physical examination by Dr. Milton I. Goldsmith revealed decreased breath sounds over the left lung with râles on both sides. Dr. Goldsmith's opinion was that there was a foreign body present. Dr. M. F. Goldsmith made excellent roentgenograms which revealed a slight shadow on the left side though it could not be stated with certainty that the shadow was produced by a foreign body. When I was talking to the patient she seemed so markedly hysterical that I felt disinclined to believe that there was any foreign body present. When, however, I examined the patient with the mirror very decided wheezing was noticeable especially on expiration, slightly at times on inspiration also. The expiratory wheeze was much more marked after expectoration.

Treatment.—The bronchoscope was passed under local anesthesia and three pieces of fish bone (Fig. 3) were removed from the left inferior lobe bronchus. The time required for the operation was three minutes and thirty seconds, exclusive of the time consumed in applying the cocain.

Comment.—The asthmatoïd wheeze was especially noticeable by mirror laryngoscopy. This is a very good way to elicit it in adults. The râles heard on both sides of the chest in this case by Dr. Goldsmith are quite common in foreign bodies lodged on one side. It seems that secretions produced by the presence of a foreign body in the bronchus of one side are aspirated into the bronchi of the other side. For this reason râles are very often not to be taken as indicative of the presence of foreign body on the side on which they are heard. Diminished air in one lung, as compared to the other, is however a localizing sign of great value.

CONCLUSIONS

1. The asthmatoïd wheeze is a sound heard by placing the ear in front of the patient's mouth during expiration. It resembles the wheezing of the asthmatic patient but is dryer. It is caused by the vibration of the air passing the foreign body in the bronchus.

2. The asthmatoïd wheeze is elicited by oral auscultation, the ear of the examiner being placed close to the open mouth of the patient. In many patients it comes out clearly during mirror laryngoscopy, especially in older children and adults.

3. The wheeze is most markedly elicited during forced and prolonged expiration. The best conception of the manner of elicitation will arise in an effort to expel the residual air from the lung. This is similar to the condition arising from a bechic

series without any interval for an inspiration to renew the pulmonary air supply between coughs.

4. The sign is of less value if negative than if positive.

5. Being heard at the open mouth, it is of no localizing value as to which lung is invaded, though there is hope that further study may develop perceptible differences between tracheally and bronchially lodged foreign bodies.

6. It is often more marked after coughing out all the secretion of which the patient can rid himself, although this is not always the case; doubtless being influenced by the size and shape of the foreign body.

7. It is of more value directly in the very recent cases. In cases of long-standing bronchitis, tuberculosis and other pulmonary lesions associated with abundant secretions there may be a wheezing audible on auscultation at the open mouth that may simulate the true asthmatoïd wheeze diagnostic of foreign body in a recent case of foreign-body lodgment.

8. The asthmatoïd wheeze was not present in any case in which a smooth rounded body was so tightly wedged in a bronchus that no air could pass it.

9. It is particularly desirable that this new physical sign of foreign body in the trachea and bronchi be tested for in every case and recorded present or absent in order statistically to determine its exact value.

10. In the author's opinion the asthmatoïd wheeze and oral auscultation are very promising additions to our diagnostic means in foreign body cases.

DISCUSSION

DR. H. H. FORBES, New York City: It seems almost futile for me to discuss this paper which is something absolutely new in the literature of bronchoscopy. I have never found Dr. Jackson wanting in any suggestion he has presented to us. We always expect something new and he has not disappointed us today. The diagnostic point is new and it will be of great use to us.

With all our efforts, Dr. Jackson goes ahead of us every time. Whenever I listen to him I get fresh stimulation for my work in this line. We ought to grasp at every diagnostic means for detecting foreign bodies. We thought some time ago that we must use general anesthesia for such work, but the more I go on with the work the more I feel Dr. Jackson is right in his position. Only recently at the Post Graduate Hospital in New York I passed the bronchoscope seven times at five and six day intervals in working out a foreign body case, and with no anesthetic.

I would make another suggestion for this work—the necessity for constant watchfulness; cases overlooked by the family physician and by the patient, who does not associate the attacks with the swallowing of a foreign body. I had three cases recently. The first patient was a woman, aged 39. Sixteen months before I saw her she had all her teeth removed

under general anesthesia. Following that she had a cough and bronchial excretion. She lost 68 pounds. She was brought to the hospital and told she would live only a few days. She coughed up a piece of bone. The family physician had not raised the question of a foreign body.

The second patient, six days after the removal of some teeth coughed up a part of the crown and the root of a tooth. He had asked to see the tooth removed at the time of operation and the dentist had said it had been "lost in the shuffle."

The third patient was a boy who had inhaled a tack. I worked over this patient seven times. The family doctor overlooked the fact that the tack was inhaled. The boy said he told him. About three months later an operation was performed for an abscess of the lung and the roentgenogram then showed the tack and it was removed only recently.

DR. E. MAYER, New York: Dr. Jackson has called attention particularly to one very important phase; it is a very easy thing to diagnose a foreign body in the bronchus when you are told the child has inhaled a foreign body; but when told there is a cough and there is nothing said about the foreign body it is a different thing, and for that reason we are especially interested in this subject. Dr. Jackson has thrown light on a case which puzzled me several years ago, for I could not understand why a general physician made a diagnosis of foreign body by auscultation, and it was there. A child about 3 years of age was brought to me because of difficulty in swallowing. Nothing was said about a foreign body and a general examination showed a swelling at the entrance of the esophagus. The child was in such poor condition that I sent him to the hospital and put him under a children's specialist, who said he heard a peculiar sound when the child belched, and it sounded like playing on a Jew's-harp very faintly, and he suggested examining with the roentgen ray to find a foreign body. It proved to be the largest I ever saw. It was a heavy metal tag used in sealing trunks, and had wire attachments and looked like a locket hanging in the pharynx. It was removed but the child died the next morning. When Dr. Jackson spoke of the peculiar sound I felt we are indebted to him for calling attention to that as an added method of examination in these important cases.

DR. E. CARMODY, Denver: This paper calls attention to two cases in the last few years with this wheeze, one a grain of corn and one a tooth. Two cases of teeth recently; one in which it was dislodged by the gag in doing a tonsillectomy and the operator did not see this tooth, but the father happened to be present and saw the tooth disappear, and he told the operator he thought the tooth had been swallowed, and it was found in the pharynx. Another case was that of a dentist extracting a third molar, which disappeared and slipped into the soft tissues. It was found by the roentgenogram in the upper jaw. These cases show how teeth may disappear. It caused a great deal of swelling in the soft tissues of the upper jaw and was apparently a disease of the parotid gland. I called in the roentgenologist. But sometimes when the grain of corn is jumping around from one position in the pharynx to another, we have to depend on the wheeze.

DR. CHEVALIER JACKSON (closing discussion): I shall not say anything further except to thank the discussers and to ask you all to test this newly discovered symptom in all cases, whether opaque to the ray or not, to get the data.

PITUITARY TUMORS FROM THE SURGICAL STANDPOINT OF THE RHINOLOGIST

OTTO J. STEIN, M.D.

CHICAGO

The rhinologist has been brought into intimate relationship with this subject, owing to a route having been selected for approaching surgically the sella, that lies within a domain in which he is thoroughly accustomed to work. In consequence thereof, a practical procedure has developed that commends this avenue of approach over that of many others.

The sellar region can be reached endonasally in several ways. The midnasal method of operating is the one followed in most of the operations. The lateral nasal method is employed in others, and a combination of the two methods is used in a few. The midnasal method follows the septum all the way through the operation. The object is to enter the sphenoid sinus and to remove its septum and that part of its upper and back wall constituting the floor of the sella on which rests the hypophyseal gland. The incision employed in this method varies considerably. Some operators, like Cushing and Kanaval, begin their incisions external to the nose. Cushing makes his beneath the upper lip, and Kanaval commences through the skin of the upper lip close to the alae nasi. These and other external methods of approach are usually employed by the general surgeon in order to secure as much room as possible, as he is not accustomed to work through smaller openings, as is the rhinologist. Again, some operators follow the methods of West and Citelli, who start far back on the septum.

With few exceptions the operators employing the midnasal or septal route make their advance between the membranes of the septum, removing the cartilage and bone as they advance all the way to the sphenoid. This is one of the desirable features of this method, as will be shown later on. Those not following this technic remove all the structures of the septum in their path. The earlier operators in this field of surgery, like Schloffer, Ollier, von Eiselberg and Proust, all of whom turned the external nose aside by various incisions, removed not only the septum but such important structures as the lower and middle turbinates, ethmoid cells, etc. West operates within the nostril and removes

the posterior part of the septum only. Hirsch, whose technic is that of the classic submucous resection method, obtains results with greater safety, less trauma, greater rapidity, quicker recovery and less untoward effects, thus bringing to the rhinologic surgeon a rational operation with which to uncover the pituitary region. In my surgical work on the hypophysis I have in all my cases followed this particular method of operating, because it appears to be the safest, most direct, and most certain and not as difficult a mode of procedure as the others.

Safety lies in confining the operation between the membranous flaps of the septum, which are brought together afterward, thus shutting off the sphenosellar area of operation from the nose entirely. The interior of the nose is more sterile than the mouth. The external nose and the nasal vestibule can be thoroughly sterilized. The endonasal route employing the midnasal method shows a minimum mortality rate over that of others, if the larger number of these operations is taken into consideration.

Statistics have a value only as far as cases are reported. Many of those in which operations are performed are not reported and their results both immediate and remote do not enter into consideration. Cope, whose compilation I report, gives a mortality rate from meningitis of 8.6 per cent. in twenty-three patients operated on by the transfrontal route, of 9 per cent. in 132 cases by midnasal method, and 25 per cent. in sixteen cases by Schloffer's method. It would seem from the foregoing that the larger mortality is due to the excessive operative work in the nose. This would apply to the lateral nasal methods, in which ethmoid exenteration is employed.

The midnasal method is not the shortest route, but nevertheless is the more direct because it has the septum as a guide, which makes the work more certain. The elevation of the membranes covering the rostrum and anterior face of the sphenoid is not as difficult as is generally believed. The utmost care should be exercised in separating this membrane around the ostea. If more room is desired, one middle turbinal should be removed. The sinus is entered at the osteum, its anterior wall is removed in all directions, and the septum between the two cells is removed without much difficulty. This is about the only place I use the chisel, unless I have one of those cases in which there is a horizontal ledge on the inner wall, to which I shall refer later. The sella fossa is best entered by using graded sizes

of sharp-bowl-shaped curets, used in the manner of a drill, after which the opening is enlarged with the mushroom head punch forceps. In this way the inferior surface of the gland is exposed to view. The capsule is then incised, the gland explored for cyst, or the tumor removed.

The field is constantly under inspection, as bleeding is not troublesome. If no tumor is found the operation can terminate as a decompression. The membranes are approximated, leaving an iodoform drain in between for sixteen hours. A wire nasal

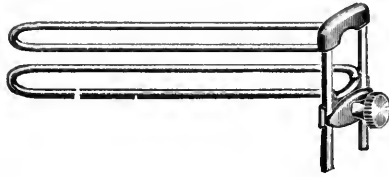
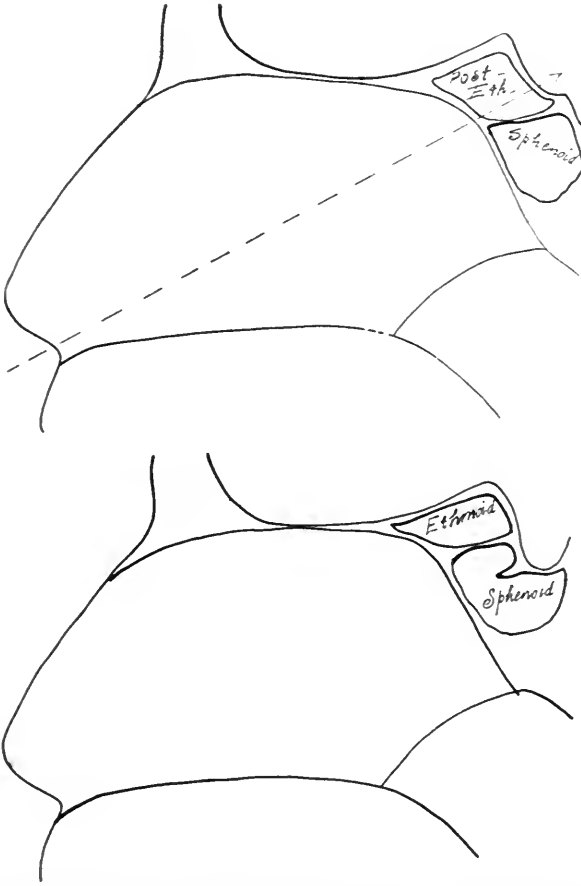


Fig. 1. Wire nasal splint which holds the flaps in approximation after the drain is removed.

splint, like the one shown in Fig. 1, holds the flaps in approximation after the drain is removed for an additional eight hours.

By the lateral nasal method of operating, the operator leaves the septum intact and approaches the sphenoid either through the nostril by removing one or both turbinates and exenterating the ethmoid labyrinth, or by making an entrance first through the maxillary antrum from the oral cavity and then into the ethmoid region. It is asserted that this latter approach is somewhat nearer, although its direction is oblique. In either instance the turbinectomy and ethmoid exenteration can be undertaken as a preliminary procedure some days before the sella operation. It suggests itself that the operator must assure himself beforehand of the presence of a sphenoid cell as well as its size and location on the side selected for operating when he uses this method. This may prove difficult for some operators, even with good pictures. If a posterior ethmoid cell should occupy the ordinary site of the sphenoid cell, embarrassment might ensue, as illustrated in Figs. 2 and 3. Besides, the cell might be so very small on the side selected as to limit one's operative approach to the sella floor. A careful study of the roentgenograms should be made in all cases, no matter what route or method of procedure is employed. This leads me to advise those taking up this work to consult the opinion of those colleagues who, working in their respective fields, may be especially informed

on pituitary disorders. Owing to the rhinologic surgeon's facility for operating in this field, the necessity has arisen for increased consideration being given to differential diagnosis and pathology. Every available bit of evidence that can contribute toward enlarging the knowledge of this subject should receive earnest attention and study. The internist or neurologist is well informed on kindred and allied conditions, therefore his advice is valuable.



Figs. 2 and 3. Showing the difficulties which might be encountered if a posterior ethmoid cell should occupy the ordinary site of the sphenoid cell.

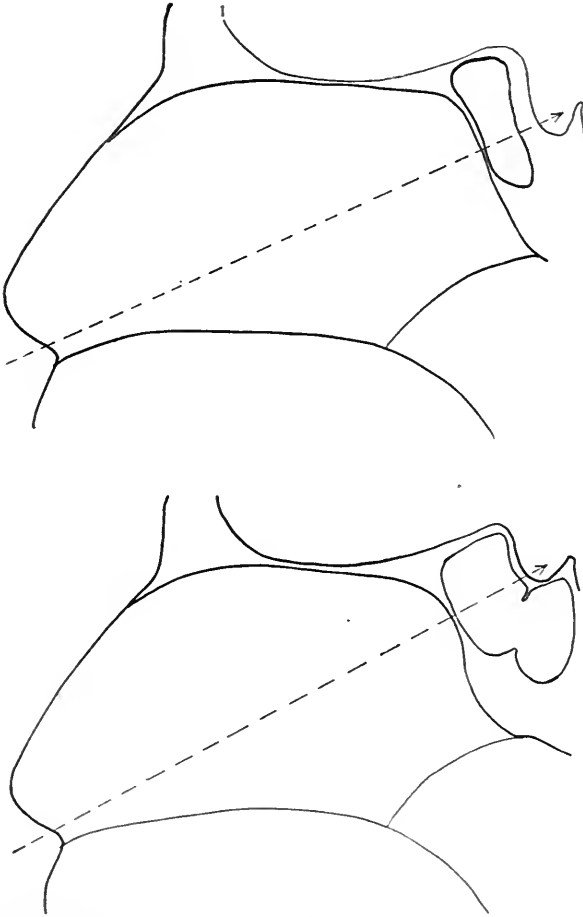
The ophthalmologist sees these patients at a period when an accurate diagnosis is most essential. By his charting of the fields and study of the discs, he may tell the location, size and extent of the brain lesion and, on account of the rapidly failing vision in many of these patients, he is the first one to have the opportunity to make the diagnosis.

The roentgenologist can be of the greatest assistance to the surgeon. His aid is greatly augmented by an intimate knowledge of this particular subject. A good picture can show evidence of capacity and dimensions, not only of the sella but also of the sphenoid cells. In acromegaly one may find enormous nasal sinuses; an increase in the size and thickness of the skull, great hypertrophy of the occipital protuberance, contraction of the orbits and narrowing of the auditory canals. The sella may be widened and deepened, and its floor may even be absent. The posterior clinoids may appear to be pushed upward, or they may be absorbed and the anterior wall of the fossa may incline greatly forward, thereby increasing the size of the diaphragmatic opening. The floor of the sella, ordinarily smooth and regularly concave, may be flattened or uneven in outline, with perhaps erosions or irregular elevations. From what has just been said regarding sellar changes, it must not be understood that all or any of these changes must necessarily be present in either acromegaly or hypophyseal dystrophy, because in acromegaly, pituitary tissue may be in the sphenoid sinus or even in the pharynx vault, and in hypophyseal dystrophy a neighborhood tumor may press from above, occasioning no further change than a sharpening of the clinoids. Infundibular tumors, like cysts, may dilate upward into third ventricle area and occasion little change in the sella. Furthermore, extrasellar pathology like hydrocephalus, syphilis, tuberculosis, etc., may create distortion of the sella, independent of pituitary changes.

In selecting a method of approach for reaching the pituitary region, we should first consider the element of safety. If to this can be added simplicity, directness, rapid convalescence and a minimum trauma, then we more nearly reach a rational procedure. None of the methods employed are ideal for all pathologic conditions. If one is satisfied with a decompression, the endonasal route approaches the ideal, but for the complete removal of a tumor, especially of considerable size, or if malignant, it falls short of the ideal. But this is true of any other route.

As one approaches the floor of the sella from below through the sphenoid sinus, a surface of about 11 by 11 mm. is presented for operation. This surface varies considerably not only in its measurements but also in its thickness. Cope took the dimensions of more than fifty skulls and found that the sella fossa, in the anteroposterior diameter, measured from 7 to 14 mm., transversely from 9 to 15 mm., and vertically from 3 to 10

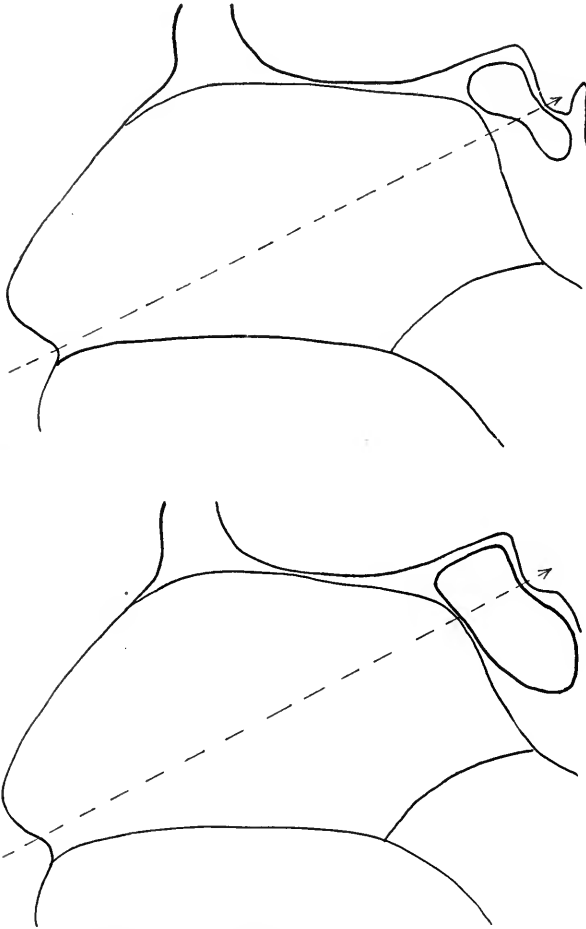
mm., while the normal gland within this cavity weighed about .5 gm. The anterior wall of the fossa inclines slightly forward and ordinarily presents a promontory on the inner surface of the sphenoid. Ordinarily the bone is thin at this place and it makes the ideal point of attack for operating, but at times it is quite thick, according to the vagaries in the development of the sphenoid sinus, as appears in Figs. 4 and 5.



Figs. 4 and 5. Vagaries in the development of the sphenoid sinus. Different thicknesses of bone are sometimes met with during an operation.

As a rule, the absorption that takes place in the body of the sphenoid in the development of the sinus commences in the presphenoid area but may be arrested here, leaving only the anterior wall of the sella separated from the sinus by a thin wall,

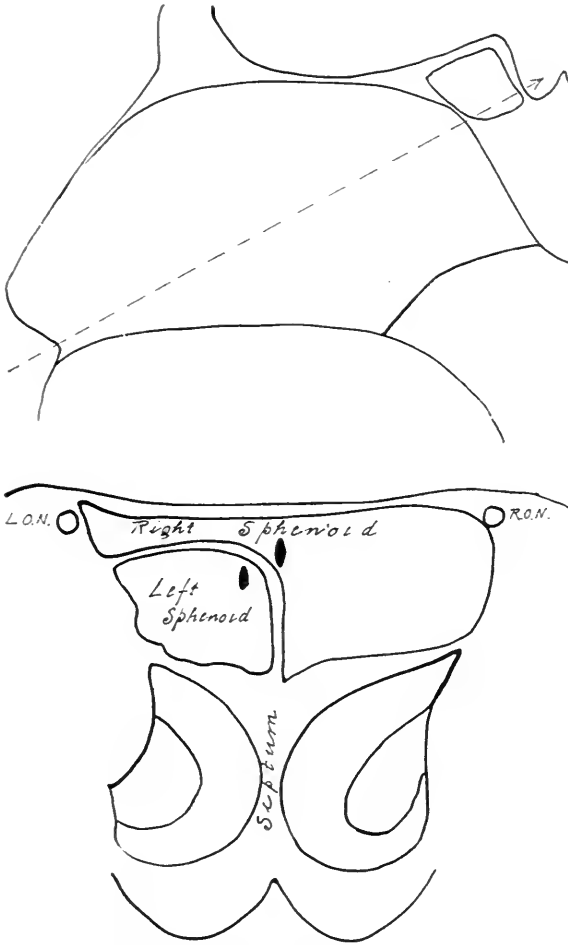
while its floor rests on solid structure as shown in Fig. 6. If the absorption proceeds normally, both the anterior wall and floor of the sella will be separated from the sinus by a thin wall. (See Fig. 7.) At the place where the arrest may occur,



Figs. 6 and 7. Showing the separation of the sinus from the sella by a thin wall of bone.

either a horizontal ridge or septum may project into the sinus (Cope). This condition, if encountered, might add some confusion and difficulty to the operation. Onodi's work on sinuses shows several variations in both position and size of the posterior ethmoid cells. In one specimen, the right sphenoid cavity occupied a part of the left cavity's position so as to exclude the latter from any relationship. (See Figs. 8 and 9.)

This condition I have myself encountered in operating for hypophyseal disease. In another specimen, the left posterior ethmoid cells occupy the position of the corresponding right

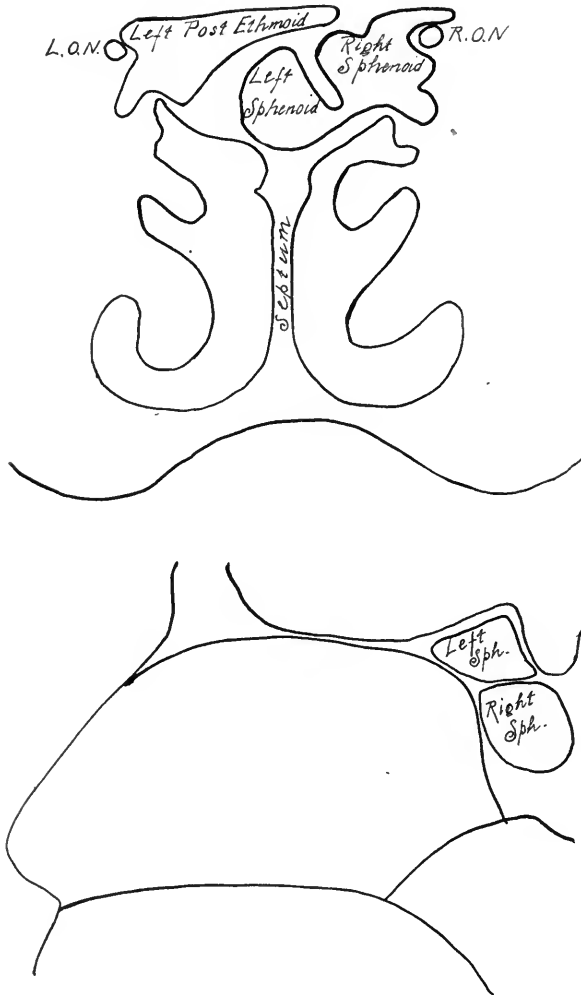


Figs. 8 and 9. Variations in the position and size of the sphenoid cavity.

sphenoid. (See Figs. 10 and 11.) When the operator is using the lateral nasal method, and especially by the oblique antrum route, these variations, if encountered, would add confusion and, perhaps, defeat a successful result.

In the performance of a number of dissections in this region I have never yet found the chiasm located in the optic groove, as stated in our anatomies. This has an important bearing on the pathology of the gland. The optic groove is a transverse

furrow about 1 cm. wide, just in front of the tuberculum sellæ, which is the upper edge of the anterior wall of the fossa. At either side it terminates at the optic foramen where the optic nerve and ophthalmic artery enter. The chiasm, I found, lies



Figs. 10 and 11. Additional variations in the positions of the ethmoid and sphenoid cavities.

just above the diaphragma sellæ, considerably posterior to the optic groove and at about the center of the fossa or even over the posterior lobe. The optic nerves pass off forward in an oblique direction. The space between the chiasm at the apex, the two optic nerves at the sides, and the tuberculum at the base

is called the trigonum presellar, and through this area passes the infundibulum, or pituitary stalk. At this place the diaphragm of the sella is thinnest and offers less resistance through the pressure of the upward growth of the tumor. From the description of this arrangement, one sees that the anterior lobe of the pituitary lies well in front of the chiasm, and tumors of this lobe may assume some size by their upward growth, before causing much pressure on the chiasm.

There seems to be considerable confusion regarding the pathologic findings in the cases in which operation is performed. It appears reasonably conclusive that changes take place in the pituitary similar to those found in the thyroid gland, of which the principal ones are hyperplasia, adenomas and cysts. All varieties of disease have been mentioned as occurring in this gland including lipoma, fibroma, cholesteatoma, echinococcus cyst, sarcoma and epithelioma (Francis Carter Wood). Primary malignancy appears to be comparatively rare while metastases have been reported. Two cases of metastasis associated with cancer of the breast are mentioned by Sckiguchi¹.

In my own cases the pathology, like that of many others, is not always quite clear. I have had nine cases that have come under my personal observation, in which all the patients except one were operated on by me. Five have been classed as adenomas and one as a cyst. Another one included in the foregoing adenoma cases had in addition a large cyst. One of the adenoma cases was pronounced by some as malignant, a parathelioma, although this patient has continually improved since he was operated in July. In two cases no tissue was removed. One of these was diagnosed as an adenoma from clinical appearances. In one the specimen was lost and the two curetfuls of tissue removed could not be identified clinically. The patients' ages ranged from 30 to 66 years. Six were men and three were women. The chief symptom in all but one was loss of vision. Three showed marked functional pituitary disturbance. Headache was present to some degree in all. In one it was very severe and almost the only symptom. The immediate result of operation was a great improvement in symptoms in five cases.

One patient died on the third day, presumably from acidosis. There was no necropsy. One patient's case was complicated by meningitis, but he survived. Another had a slight hemorrhage in the left hemisphere, but completely recovered in a few days.

1. *Annals of Surgery*, 1916, **63**, 297.

The remote effects from operation after one and one-half years show one death from a cyst that was apparently not present at the time the operation was performed, when a large decompression was done for an adenoma. There was a disappearance of the headache in most cases, also the vertigo, when present. Continued improvement resulted in the restoration of sight, or the improvement was maintained in those patients showing immediate benefits.

I have presented at this time only the salient features of these cases because some of them have already been reported by me, and others are too recent to give remote results. I shall therefore consider it a privilege if I may be permitted to make a further report at a future time.

ASTHMA IN A WIDER ASPECT

HARRY L. POLLOCK, M.D.

CHICAGO

It is a well known fact, that in any disease in which the etiology is not clearly established or is unknown, more papers are presented than on one which is well understood, each author in turn giving his deductions and conclusions drawn therefrom. This is especially true of a disease which affects a large number of individuals and which is classed under the title of one of the commoner diseases.

This is certainly clearly established in the affection under consideration, namely, bronchial asthma. In reviewing the literature one is amazed and stupefied by the vast amount that has been written. Dr. Orville H. Brown¹ in his monograph "Asthma" published this year, 1917, gives 993 references to articles previously written, which does not nearly cover the literature on this subject, as I have satisfied myself by the perusal of the literature.

That so much has been written is perhaps due to the fact that up to the present time, no definite etiology has been established and consequently each individual has the prerogative of theorizing and bringing forth arguments to sustain his hypothesis.

My sole aim in presenting this subject is to attempt to gather the most salient points in this voluminous chaotic mass of words and present them in a tangible form, so that we may have some rational form of treatment to alleviate the dreadful condition of these victims.

That the treatment of asthma belongs primarily to the rhinolaryngologist is or should be fairly well understood by all, for the main attack lies within our jurisdiction, but at times it is necessary to call the internist to work conjointly with us.

ETIOLOGY

That the severe acute attack of asthma is caused by a spasm of the musculature of the smaller bronchi is, I believe, accepted by all. This fact and this alone is the only one in which we all agree. The explanations of how this etiologic factor causes

1. Brown, Orville H.: Asthma 1917.

this phenomenon are as varied as are the writers on the subject. For the past several years Dr. J. C. Beck and myself have presented several papers touching on this subject, and the more we study, and the more experience we have had in the examinations and treatment of these patients, the more convinced we are that the underlying etiologic factors can be traced to a disharmony of the ductless glands. What the disturbing factors causing this disharmony may be, are still a matter of conjecture but we feel that we are on the right track and will eventually be able to throw some definite light on the subject.

That the spasm of the bronchi is due to an irritation or stimulation of the vagi is now also accepted by nearly every one; as experimentally the same syndrome of asthma can be caused in animals by a mechanico-electrical stimulation of the vagus.

We know that many cases of asthma are associated with some pathologic conditions of the upper respiratory tract, such as suppurative ethmoiditis with or without polypi formation; hyperplastic ethmoiditis or a suppuration of the antrum or sphenoid. Yet that this pathologic condition is not the underlying factor is proven by the large number of patients who give evidence of these conditions without showing the slightest symptoms of asthma. These conditions certainly play an important rôle as an exciting cause, because after having these pathologic conditions cleared up all cases are benefited to a greater or less degree. I have yet failed to see one whose asthma became aggravated by surgical intervention on the sinuses.

In many of these cases treated by the general practitioner, the condition of hyperplastic ethmoiditis especially, is overlooked, as it is not easily diagnosed and consequently many patients are reported as being free from any pathology of the upper respiratory tracts.

Last year before this Society, I gave our theory as to the cause of various diseases, such as otosclerosis, ozena, osteomalacia, asthma, etc., and attempted by deductions and actual microscopic pathologic findings to prove that they all were dependent on some change in the ductless glands. The exciting causes in the different diseases are varied, but the underlying etiologic factors are a change somewhere in the system of the ductless glands.

At this point I desire to quote some abstracts from an article by Dr. Ernest Zueblin².

2. Zueblin, Ernest: *Med. Rec.*, New York, 1917, **91**, 364.

"As the anatomical basis of bronchial asthma we admit:

1. A spasmodic contraction of the muscle fibers of the bronchi, arising by direct or indirect influences.
2. A swelling of the bronchial mucous membrane, a fluctuating turgescence. A diffuse hyperemic swelling.
3. A bronchiolitis exudativa.
4. Nervous impulses on the pneumogastric nerve and vaso-motor influence."

Further on, the same author states that according to more recent conception the vagal center is influenced by impulses that depend on the secretions of the posterior part of the pituitary body. This complex organ containing many different nerve cells not only is supposed to be the receptor of all of the common sensibility but also all coordinate involuntary motor impulses are thought to arise from this organ. Furthermore, the pituitary vagal center is made the terminal of sensory impulses from the nose. Such a fact has been brought out by Cyon's experiments demonstrating that the destruction of the pituitary body completely destroys the reflex sensibility of the nasal mucosa. Through the vagus efferent nerves, motor impulses are transmitted to the lungs and so a complete reflex arc is established. The predisposing cause of bronchial asthma is attributed to a hypersensitiveness in the posterior pituitary body, which may arise from the irritation of the bronchial mucous membrane by some substances contained in the inhaled air or substances arising from toxic or other products of imperfect catabolism. These substances are thought irritating for the bronchial sensory and organs of the vagus. Under normal conditions the afferent impulses from the bronchi, reaching the vagal centers evoke just sufficient efferent motor impulses as to create periodic contractions of the bronchi and secretions of mucous, to promote the activity of the ciliated epithelium and to insure elimination of the poisonous waste products. When, however, a hypersensitiveness of the vagal center exists, excessively violent stimuli are sent in the direction of the bronchial muscles and hence muscular constriction of the bronchi, swelling of the mucosa, and broncho-stenosis takes place and asthma is the consequence.

This explanation in my judgment covers the very groundwork of the etiology of this disease, and most of the previous theories while not complete tend to prove more emphatically the above deductions.

It is but a short time since Vaughan first gave us his work on split protein poisoning as the causative agent of many of

these heretofore unexplained phenomena. It is easy to comprehend that the poisons arising from the split proteins may act as an exciting factor in asthma by causing an irritation of the posterior lobe of the pituitary body or the bronchial sensory end organs of the vagus, thus creating the phenomena of asthma.

SUGGESTIONS AS TO TREATMENT

Dr. I. Chandler Walker³ of Boston has recently presented his fifteenth study on asthma. If we follow his work carefully we find that he like most investigators attempts to isolate the exciting cause, and avoids ascertaining the underlying or predisposing pathology. His studies are profound and of inestimable value and are well worth scrutinizing. He attempts desensitizing the patient to the protein, pollen, dust, etc., to which they are sensitive.

He determines the exciting cause by subjecting the patient to vaccination with various substances and ascertaining the one to which the patient reacts.

In those cases in which there is no reaction to any of the proteins, pollen or dust from animals, he tests the blood for agglutination with the staph. pyogenes albus. If such cases show agglutination he vaccinates them with increasing doses of a stock vaccine of pyogenes albus. If it does not agglutinate, he isolates a diphtheroid bacillus from the sputum and uses a vaccine of this bacillus. Many experimenters have obtained good results in these cases of inoculations of proteins, and explain the etiology as being due to sensitization, clearly overlooking the fact that these agents are only exciting and not predisposing factors. As just explained, it is possible that these substances, pollens, proteins, etc., act on and irritate the bronchial sensory end organs of the vagus and thus cause the efferent motor impulses which create these powerful stimulations to the bronchial muscles and mucous membrane, and produce the syndrome of asthma. Inversely, by not permitting the formation of these protein poisons or by not allowing the pollens and dust to come into contact with the bronchial mucous membrane, asthmatic attacks can be prevented.

The desensitization of these individuals against their special sensitizing agent, no doubt keeps the bronchial mucous membrane from becoming irritated and thus in turn prevents the efferent vagal stimuli from reaching the musculature of the small bronchi.

3. Walker, I. Chandler: Jour. Med. Research, September, 1917.

Another partially successful line of treatment is that which was first suggested by A. Ephraim in 1910⁴.

This consists of endobronchial applications of medicaments directed to the mucous membrane by the use of the bronchoscope. He found that a novocain-adrenalin solution gave him excellent results. Various other remedies were employed, but not with the same degree of success. He observed during an attack of asthma that the bronchial mucous membrane was red and swollen and a great deal of thick tenacious mucus was present. Immediately after this endobronchial treatment with novocain-adrenalin solution by the aid of the bronschoscope, the patient experienced more difficulty in breathing but this was only transitory for soon there was an expectoration of a great abundance of thin watery secretion and soon following this great relief was obtained. Whether the relief was obtained by the application of the solution or by the mere mechanical irritation of the tube, was not clearly understood, but I am confident it was due to the former, as we have suspended patients in direct laryngoscopy and instilled a solution of cocain and adrenalin and obtained similar results, so that the theory of direct irritation from the tube can be quite reasonably presumed to be incorrect. In many of Ephraim's cases only one treatment was necessary to obtain from six to nine months' relief; in others it required three to four treatments, and in a very small number repeated applications direct to the mucous membrane failed to give any relief.

One of the latest lines of attack in asthma is that which was recently reported by Kahn and Emsinger⁵.

This treatment consists of injection of autogenous defibrinated blood. Their theory of the etiology is based on the supposition that the attack is due to anaphylaxis. As their deductions are very plausible I take the liberty of quoting the following:

1. Asthma is due to a spasm of the smaller bronchi.
2. Spasm of the bronchi is a manifestation of anaphylaxis.
3. The anaphylactic phenomena may be explained on the basis of protein sensitization.
4. Whether the protein gains access to the body by the nasopharynx, by the intestinal tract, by the respiratory system or by some other portal, it is probably absorbed by the blood, if so it should be found in the blood especially just prior to or during an attack of asthma.

4. Ephraim, A.: Berl. klin. Wehnschr., 1910, **47**, 1276.

5. Kahn, M. H., and Emsheimer, H. W.: Arch. Int. Med., 1916, **17**, 455.

5. The rational method of active immunization in anaphylaxis consists in repeated injections of the causal protein. If the previous premises are true in asthma, immunization by repeated parenteral injections of autogenous defibrinated blood obtained during an attack should be beneficial."

The process consists in withdrawing from 20 to 30 c.c. of blood and defibrinating same as it is withdrawn. It is immediately reinjected subcutaneously and this process repeated weekly until about ten injections are given. They do not explain, however, why results are obtained by reinjecting this small amount of protein, while the whole circulating system contains vast quantities of the same protein.

The last case which they reported was in a woman who stated that whenever she became pregnant the attacks of asthma never occurred. In the course of their treatment she became pregnant again and naturally they refrained from further treatment and the asthma did not recur. This case is of extreme importance to me, as we know that during pregnancy there is an increase in size of the pituitary body and supposedly an increase of the secretion from the gland is thrown into the circulation. Is it not then probable that this overstimulation of the gland prevents the recurrence of the asthmatic attack? In a recent case which I treated by the defibrinated blood therapy, there was a marked exacerbation of the attack, which would not yield to adrenalin, but did to one injection of pituitrin, which acted almost as a specific. As far as the action of a hypodermic injection of adrenalin is concerned, it is entirely useless for me to state, as you well know, that usually one injection of from 10 to 15 minims stops the attack immediately. I have found, however, that if it is given with equal amount of physiologic sodium chlorid solution, the action is more efficacious and of longer duration. At this point I desire to call your attention to the foolhardy practice of many physicians of placing adrenalin and the hypodermic syringe at the disposal of patients, as they very rapidly acquire the adrenalin habit. I have under observation a patient that I found using hypodermatically as much as 1 ounce per day of the 1:1,000 solution. I have seen others who were injecting as high as 20 minims every two to three hours. This adrenalin habit is much more difficult to combat than is the morphin habit, as the patients crave and beg for it and become desperate if it is withheld.

In most patients I have found it extremely difficult to break

them of their desire for adrenalin and permit me to put them on any other line of treatment.

As many patients obtain beneficial results from these various enumerated methods of treatment, it is my custom to give them the benefit of a systematic course, one after the other, until the patient has had beneficial results or until all known methods have been exhausted.

TREATMENT EMPLOYED BY THE AUTHOR

I shall now outline my course of treatment in these cases. If the patient is first seen during an acute attack my aim is to give him immediate relief as that is what he seeks. A hypodermic injection of adrenalin from 10 to 15 minims is given and the same dose repeated when necessary, often at intervals of from two to three hours. Then the patient is thoroughly examined, also going into the history and laboratory and physical findings. The bowels are cleaned out thoroughly and a diet is given as free from proteins as possible. The patient is kept at rest away from the noise and dust. Usually in the course of a few days the acute attack has subsided and then we are ready for the surgical interference of the upper respiratory tract, providing a pathologic condition is found. As previously stated most of the patients give evidence of some pathology in this region, either suppurative or nonsuppurative and this must be corrected as far as possible. The ethmoids are exenterated and the other sinuses are explored for whatever is necessary must be done thoroughly. In the meantime the excretions and secretions are given proper attention and the diet previously outlined is adhered to. Often during this part of the treatment it is necessary to resort to the use of the ductless gland products, either adrenalin or pituitrin. I have the internist always associated in the treatment and management of these cases.

If the patient does not give any evidence of pathology in the upper respiratory tract, I then turn to the internist for aid. The patient has a thorough physical examination made, and if any foci of focal infection are found, such as a chronic appendicitis, a cholecystitis, or similar infective conditions, these foci are eradicated. If nothing is found I proceed to further treatment.

The patient is then given a systematic treatment of the application of cocain and adrenalin by the bronchoscope or suspension laryngoscopy. The latter procedure is employed more often as a better view of the larynx and trachea is obtained. It is not

difficult to pass the bronchoscopic tube while the patient is suspended and make application direct to the bronchi.

Since studying the reports of the treatment by reinjection of defibrinated blood, in conjunction with our internist, I have treated a number of cases by this method, but my results were much different than those of the original investigators. They report that they had no immediate bad results from the injection, but in several of my patients I obtained an anaphylactic reaction, resulting in profound cyanosis coming on within an hour after injection. In one of these, one dose of pituitrin cut short the attack at once. I have for the present discontinued this line of treatment.

CONCLUSIONS

1. The underlying causative factor of asthma is undoubtedly due to a disharmony in the ductless gland system.
2. The exciting factor varies. It may be dust, pollens, excreta from animals, split proteins, poisoning, etc., but unless there exists a disharmony in the ductless gland system asthma cannot result.
3. A systematic line of treatment such as is suggested in this paper will give a beneficial result.
4. To attempt a permanent cure, we must study our ductless gland system more thoroughly and that will come as soon as the physiologic chemists will have proved such conditions by experiments. All we can do as clinicians is to examine our cases carefully and treat them on a rational but at present empirical basis. The reports should always be as enthusiastic as possible yet absolutely truthful.

DISCUSSION

DR. JOSEPH C. BECK, Chicago: This subject of asthma I am sure is of great interest to all of us. I have repeatedly said that I have never seen a case of asthma that was cured by nasal operations, and I am still of that opinion. By cure I mean no recurrence for at least five years. That period is taken by the majority of the public health authorities as required for a cure of carcinoma or any disease that is deeply rooted. Now the cures we read about in textbooks or journals following nasal operations are of much shorter duration. I would like to hear in this discussion reports of cases of true asthma that have been cured for five years or even approaching that time. Dr. Pollock has outlined the course of treatment we try to follow until the patients are relieved or until we give up treatment. I should say that in the majority of them we do not give up the treatment; the patients give it up. There is not a great deal of benefit in many of these cases and it is discouraging to me, indeed, in so far as any curative result is concerned. The point on the internal secretions, the question of the etiology and the treatment by that method, appeals to me more than any other, because

I find in the study of pathologic changes of the bones there are changes which are very significant in cases of asthma. If you examine the turbinates and septums that are removed for the condition of asthma, you will find a definite rarefying process in the bone which is analogous to changes in the osseous structures that are due to disturbances of the hypophysis and adrenal glands. The deductions drawn by men who have studied the matter is very striking and very encouraging. We should by means of pituitary and adrenal gland feeding, perhaps better than by injection of adrenalin or pituitary accomplish better results. But until we can prove by blood examinations which of the internal secretory glands is deficient or excessive in the system we are not going to make much headway. I might say that at the Mayo Foundation they have isolated a substance so far as the thyroid is concerned. Thus far it is believed that some of the disturbances of the glands of internal secretion are due to some protein poison. Vaughan and others have worked along this line. They are supposed to be bacterial products which are constantly being absorbed into the system from the gastro-intestinal tract and other foci of chronic infection. Therefore the asthma cases should receive the greatest attention in respect to the gastro-intestinal tract and other foci of infection. We have held that a great deal of this infection comes from sinuses and teeth and we must stick to that. I believe in every case of chronic asthma we should remove the tonsils almost on suspicion. In employing the bronchoscope the improvement is definite and I think it is due to the application of the medicine rather than the bronchoscope. Sometimes the slightest attention to the nose such as the prying off the middle turbinate or opening up the ethmoid cells, produces a good effect, but it is usually temporary.

DR. R. E. MERCER, Detroit: In the past two years, I have been doing quite a little work along this line and with some rather curious results. For the foods, we first took the method of Goodale. For the bacterial group, we have taken the different organisms, grown them on bouillon and, by the method of Ferry, made a paste which is very convenient to use.

We have had many patients in whom the reaction has been marked. In these, by desensitizing, we have stopped the asthma. I do not mean that we can cure every case. We found a good many cases in which different foods have been the cause, and one of the curious things has been that barley has been one of the most common, possibly because of the use of barley water in gastro-enteritis in children.

In sensitization the specificity has been called into question, one will get many cases which will show sensitization to the *Staphylococcus albus* and not to the *aureus*, to the pollen of goldenrod and not to ragweed, that is, to the weaker and not the stronger members of the groups.

About nasal discharges, we are stopping absorption of proteins and probably bacteria that have been penned up in the nose, when we promote nasal drainage by operative work. The same with the tonsils. Dr. Pollock speaks of basic disharmony. I think that he is getting at the thing from the right standpoint. The results I have had are rather contradictory. We have had cures, I cannot say for five years because we have not been doing it that long. Take a group of cases which show sensitization to everything and you cannot do anything by desensitizing methods, as far as my experience goes.

DR. KATE W. BALDWIN, Philadelphia: As to cases that respond to intranasal operations, I have at least one case—that of a young man who has not had asthma for four years. The only treatment after having

suffered for ten or fifteen years was an intranasal operation and a little calcium iodid.

DR. POLLOCK (closing discussion): There is nothing further except, as I stated, all these known methods seem to give results. They have reported cases as cured. In one series of five cases with defibrinated blood, all were apparently cured. I have seen cases treated by sinus operations and fed on a gland diet, and they go along several months practically free. When a change of weather comes, the bronchial mucous membrane becomes irritated and then there is a recurrence of a slight attack, and these are the cases reported as cured. They are only relieved, not cured. I remember one case, a dentist, who was unable to perform his work on account of the asthma. He had a distinct hyperplastic ethmoiditis. The sinuses were completely cleaned out and a vaccine made at the time. I think it was a staphylococcus infection and immediately after the operation we gave him the vaccine for two months. At the end of that time he stated he was cured. He went back to his usual work. Just about this time he was exposed to a severe rain storm which caused no symptoms. Two months after that he had a slight attack of asthma again, but so slight he was able to take care of his work. There is no question that if you take these patients and clean out their sinuses first and put them on a rational form of treatment you will get relief in a great many cases, but I myself have failed to see any case that was free from asthma for a period of from four to five years. As I stated before, this desensitization is only palliative treatment. It is not treating the underlying factor. When physiologic chemists can say this or that individual has too much or too little thyroid or hypophysis secretion then will we be able to say we have a cure and not until then.

THE ETIOLOGIC RELATIONSHIP OF INFECTED FAUCIAL TONSILS TO CHOREA

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In the course of our work in the Baird-Dulaney Hospital, we have for the last few years been particularly interested in the subject of focal infection in general. By means of a close examination and study of individual cases, and instituting systematic efforts to locate foci of infection, we have been able in a large number of cases to clear up what appeared at first to be very obscure conditions. The subject of focal infection in all its aspects is a broad and fertile one, and furnishes a field for very interesting and profitable study. While we realize clearly the limitations of focal infections as etiologic factors in disease, and have seen that there is undue and even harmful enthusiasm shown on the subject by some men, still we have demonstrated to our own satisfaction that focal infections play a large part in the production of many human ills. The literature on the subject is increasing every day, and new evidence is constantly being added to the already large amount bearing on the subject.

Having on one occasion removed some badly infected tonsils from a patient having a mild form of chorea, and noting an immediate and surprising relief of choreic symptoms, we determined to study chorea on the hypothesis that it was a nervous manifestation secondary to some primary focus of infection. Since then we have treated a limited number of cases of chorea and allied conditions by removing infected tonsils, and have observed some of them as long as three years after such treatment, and our results in these cases have justified us in making this preliminary report on the subject. Let us say here that we realize that our series of cases is small, and the amount of experimental work is meager, therefore we do not wish to state any final conclusions, or make any positive claims. We merely present the results of our work as far as we have gone with it, for what it may be worth.

In the etiology of chorea, very little definite information is to be found. The theories advanced concerning its causation are conflicting and purely speculative. But all the authors are in

agreement, in that chorea is associated in the large majority of cases with some evidences of a systemic infection. Dieluafoy in his text makes particular mention of the occurrence of chorea with endocarditis. Strumpell, Anders, and Osler mention the association of endocarditis, pericarditis, and rheumatic fever with chorea.

Holt cites the claim of some authors that it is due to a reflex irritation from adenoids and enlarged tonsils, and states that the association of chorea and enlarged tonsils and adenoids is not infrequent. Some of the later authors ascribe an infectious origin to chorea, and Matthews in 1916 reported four cases of chorea treated at the Mayo Clinic by the removal of diseased tonsils, with relief in three of the cases. Other etiologic factors mentioned by different authors are: fright, overpressure in school, anemia, chronic malarial poisoning, phimosis, intestinal parasites, delayed menstruation, and ocular defects.

The number of cases constituting the basis of this report is fourteen. The ages range from 3 to 35 years. Four of the cases were adults, and of these adults, 75 per cent. were females. Ten of the cases occurred in children, and of these, 60 per cent. were females.

The nervous symptoms shown by these patients were mostly motor, and displayed derangements extending in character and severity from spasmodic twitching and fibrillation of small groups of muscles, to the irregular, jerky, semipurposful movements of all the muscles of the skeleton. In a particularly severe case, partial incoordination of all the skeletal muscles was observed in addition to the jerky motions. Poor and disturbed sleep was a noticeable feature of all the cases. The motor disturbance extended to the muscles of articulation in the majority of cases. In some of the adult cases a certain amount of mental derangement was present, either taking the form of hallucinations of persecution and other morbid notions, or merely paroxysms of hysteric crying.

Almost without exception all the patients gave a history of previous tonsillitis, and the adults a history of repeated attacks extending over a period of years. Some of them stated that they had not been troubled with local symptoms from their tonsils for some months previous to the beginning of their nervous troubles, with the exception probably of a sensation of slight irritation in the throat, entailing a "clearing up" process in the morning.

ASSOCIATED CONDITIONS

All the cases except one showed some evidence of cardiac damage, generally taking the form of endocarditis. This was evidenced by signs ranging from slight, barely audible roughened valvular sounds to frank endocarditic murmurs.

Rheumatism played a part in the histories of most of the patients. One of the patients had subacute arthritis at the time she came under our care. One of the men gave a history of acute polyarthritis some months before the onset of his nervous symptoms. Four of the patients gave a history of acute recurring arthritis previous to the appearance of nervous symptoms.

Fever of a greater or less extent was an accompaniment of all the cases except two when they came under our observation. The temperature in no instance was above 101 F. The two exceptions occurred in adults, and in these the temperature was constantly slightly subnormal, but more noticeably so in the early morning.

The diagnosis of these cases as nervous conditions secondary to primary foci of infection was made only after careful and repeated examinations of the patients. Thorough physical examinations were made in an effort to find other etiologic factors if possible, and we did not lay the blame on the diseased tonsils as being the cause of the trouble until we had eliminated other causes. Careful laboratory examinations were made in every case. In three of the cases Wassermann tests were made on the blood, as they were the only ones which presented anything in the history or findings that made us at all suspicious of syphilis. In these cases the reaction was negative.

The acute cases showed a polynuclear leukocytosis of greater or less extent. The blood was cultured from these cases, and a growth obtained in only two. One of these presented a growth of a short-chained, nonhemolyzing streptococcus, 0.5 c.c. of a bouillon culture of which did not cause death after injection of the peritoneal cavity of a guinea-pig. No changes of importance were observed in the pigs thus injected. Animal experimentation was not systematically or extensively carried out in this series. Cultures made of the tonsils after their removal gave growths of streptococci, staphylococci, and pneumococci, together with other organisms commonly found in the oral cavity. No one organism was noted to appear more constantly than any other, or to appear the predominating organism, so we have

made no attempt to ascribe any degree of specificity to any of the organisms.

The tonsils in most of these cases had very much a similar appearance of those described by Ophuls which he found to contain the foci of infection in cases of nephritis. The majority of them were not enlarged, but on the contrary were rather shrunken. In a superficial examination of the throat, the average clinician would call a large number of them normal, or only slightly diseased. They were of the type that we speak of as being "submerged." The surface was grayish-white, showing cicatrization, and the openings of the crypts were quite narrowed, and sometimes apparently obliterated. The pillars are often bound tightly to the tonsils with thick heavy bands. A section of such a tonsil shows bands of fibrous tissue throughout the tonsil, the contraction of which has caused a stenosis of the orifices of the crypts. This occlusion of the orifices has caused the crypts to become distended and retain the pus, forming "pus pockets" and furnishing reservoirs from which infection can constantly be absorbed.

An excellent way to diagnose these tonsils while still in the throat, and a way to absolutely place the blame on them as containing pus pockets, is to use the small cold electric light on the DeZeng-Standard diagnostic set. This light, white and brilliant, but slightly larger than the head of a match, is placed in firm contact with the lower border of the tonsil. Thus the tonsil is literally transilluminated. The tonsil such as has been described appears pale, translucent, and shows dark areas which are the pus filled crypts. Tonsils of this kind are the result of many repeated attacks of acute tonsillitis of greater or less severity, which has caused a process of slow cicatrization to take place, producing adhesions to the pillars, and occlusion of the orifices of the crypts preventing free drainage.

TREATMENT

The treatment of these cases consisted first in the removal of the tonsils, and when indicated, the adenoids. This we did under general anesthesia, using blunt dissection to free the pillars, and the snare for enucleation. After the hemorrhage was controlled, and we were certain that the tonsil was removed in its entirety with the capsule, we applied half-strength tincture iodine to the fossae. Daily cleansing treatments were made for some time after the enucleation.

In some cases as a result of long continued infection in the

tonsils, other tissues of the nasopharyngeal region had become infected, such as adenoids, enlarged turbinates, etc. These fields, when they existed, were cleaned up as part of the treatment. A thorough search was made in every case to be sure that no other infected focus remained.

As in all cases of focal infection from whatever cause, we insist on adequate after-treatment. This consists in the main of thorough intestinal elimination, mercury, arsenic, potassium iodid, strychnin, the salicylates, or other medication as the indications may be. In most of the cases proper hygienic management with rest was all that was necessary.

RESULTS

The treatment as outlined above resulted in the complete relief of the nervous symptoms, and a marked improvement in the general condition of the patient. In only one case were we denied the removal of the tonsils. This case improved greatly under local treatment of the tonsils and the use of autogenous vaccines made from the bacteria recovered from the tonsillar pus. The patient became free from choreic symptoms, but a condition of nervous irritability still persists, and a normal condition has never been reached.

REPORT OF CASES

CASE 1.—L. F., boy, aged 15, was referred to one of us for the correction of errors of refraction, to which his nervous symptoms had been attributed.

History.—The boy's parents stated that he had been of a nervous temperament since early childhood, and had never been able to make the progress in school that he should. An oculist in another city had corrected his errors of refraction, to relieve a spasmodic winking of his eyelids, and a circumcision for the relief of other minor nervous symptoms. For awhile the boy was apparently better. It was learned that he had repeated attacks of tonsillitis in early childhood, but for some time previous there had been no local symptoms beyond those of a slight irritation. With the exception of measles, the history was negative as regards infections. For a week before we saw him, parents noticed that in performing a voluntary movement, his motions were jerky and irregular. These choreic motions rapidly grew worse and more extensive, and soon involved all the skeletal muscles. At the beginning of his nervous disturbance, he began to sleep poorly, and soon showed marked insomnia.

Examination.—On examination the patient appeared an undernourished, enemic boy. While observing him when seated, any number and variety of spasmodic muscular movements were to be seen. They were general and affected all the muscles, and were

more pronounced on the left side. The majority of these movements seemed to have their beginning as purposeful movements, but terminated in the incoordinated jerk of chorea. In attempting to pass through a door, he would show a marked incoordination of his lower extremities, and his gait was hesitating, jerky, and uncertain. This muscular incoordination extended even to the eye muscles, as indicated by an extreme difficulty in focusing an object. He attempted to close his left eye in order to get a clear view of an object with his right eye, and vice versa. It was impossible for him to read the test type at any distance on account of a spasmodic contraction of the muscles of the left side of his neck, which would, after making a few attempts to read the type, draw his head over on his shoulder. Physical examination showed nothing remarkable beyond the points already mentioned, with the exception of a slight roughening of the valve sounds. Temperature 99.4 F., pulse 100. Laboratory examination revealed nothing beyond a low grade secondary anemia, and the leukocyte count was 10,000. The Wassermann and tuberculin reactions were negative. His throat showed submerged, adherent, cicatrized tonsils. On firm pressure inspissated pus was expressed from the apparently obliterated orifices of the crypts. Culture of this pus gave growths of a short-chained nonhemolyzing streptococcus, with a few colonies of a staphylococcus, and a diplococcus resembling the pneumococcus. Cultures taken from the tonsils after removal gave the same organisms. On "trans-illuminating" the tonsils they had very much the same appearance that we have previously described.

After a week of observation and careful study of the patient, we concluded his case to be one of focal infection, with the tonsils harboring the focus. The tonsils and adenoids were removed under general anesthesia.

Outcome.—Twenty-four hours after removal of the tonsils, the extreme muscular spasm and incoordination began to improve, and in a week he was entirely free from them. However, a condition of nervous irritability remained, but no more than had been noticed for some years, and this gradually improved. His general physical condition was improved in every respect, gaining remarkably in weight. He is now in school, and his teacher reports that his progress is satisfactory.

CASE 2.—L. B., unmarried woman, aged 21, admitted to hospital Dec. 5, 1916, referred by Dr. J. L. W. She complained of convulsive movements of the muscles of the left shoulder of such severity as to jerk the entire left side nearly free of the bed at times, and which required chloroform for control.

History.—Beyond the usual diseases of childhood the patient had always had excellent health, but had suffered at intervals from mild attacks of acute tonsillitis, but at present the tonsils were not causing any local symptoms. Ten months previously she had an attack of la grippe, and from that time on the patient had noticed a condition of nervous irritability. Anything that startled her would cause a convulsive twitching of certain groups

of muscles, and she would "lose control of herself." This condition grew gradually worse, accompanied by considerable loss in weight. For a month before we saw her these attacks became more severe and appeared without any provocative influence, and began to appear twice a day and oftener. A week before entering the hospital she was confined to the bed, and for the five days previous to her entrance, chloroform was required to control these convulsive seizures.

Examination.—The girl was anemic with evidences of a loss in weight. Observing the patient during one of the attacks of muscular spasm, there was seen a peculiar jerky, spasmodic movement of the left shoulder muscles, which jerked it antero-posteriorly; and so extensive and violent were these movements, that it looked at times as though she were going to jerk herself off the bed. These attacks appeared any number of times a day and without any apparent provocation, and were not accompanied by any loss of consciousness. Any attempt at voluntary movements would precipitate an attack.

A general physical examination failed to reveal anything remarkable beyond pathology in the nasopharyngeal region. Temperature 99.2 F., pulse 95. Slight roughening of the valve sound at the apex. The throat showed moderately enlarged, firmly adherent, submerged tonsils, with the peculiar grayish-white surface. At the upper pole of the right tonsil there was an opening communicating with the interior of the tonsil, from which pus could be expressed. The tonsil was literally a pus sac. There was present in this patient a condition which I have observed in cases in which there has been a tonsillar infection of long standing. This consists of patches of lymph follicles which have become hypertrophied and by coalescence have produced a raised granular patch on the posterior pharyngeal wall, and on the upper part of the fauces. Both middle and inferior turbinate bones were enlarged, turgescient, and showed a rough granular surface. In the right nasal fossa was found a pedunculated polypus the size of a hazel nut.

Treatment.—The tonsils were removed under general anesthesia with the snare. Pus exuded in large quantities. The fossae were swabbed with half strength tincture iodine. The turbinates and polypus were removed under local anesthesia later.

Outcome.—There were no convulsive seizures of the muscles at any time following the removal of the tonsils. The nervous irritability was still present to a marked degree, but was alleviated by the exhibition of bromids, which had shown no effect before the removal of the tonsils. This irritability gradually cleared up, and in a few weeks had completely disappeared. Six weeks after leaving the hospital she gained 20 pounds. At the present the patient is entirely free from symptoms, and states that she feels as well as ever. She has a good color, sleeps well, and presents as near normal condition as possible.

SUMMARY

1. All the cases of chorea and choreiform affections forming the basis of this report appeared to be secondary to primary foci of infection which were found in the tonsils.

2. The tonsils in these cases showed the results of many attacks of tonsillitis, and at the time of removal were in an infected condition.

3. The organisms harbored by the tonsils were the types of organisms commonly found in tonsillar infection, but the streptococcus and pneumococcus groups were apparently the most constant.

4. The diagnosis of a nervous condition secondary to infection elsewhere in the body was only made after repeated examinations and a study of the case.

5. The removal of the tonsils together with any other infected areas in the nose or throat, gave complete and immediate relief which has lasted in all the cases until the present time. Some of these cases have been observed for three years after the disappearance of the nervous symptoms.

CONCLUSIONS

1. There are a large number of cases of chorea and choreiform affections which are secondary to primary foci of infection.

2. Only after the most thorough and repeated physical examination and laboratory investigation should the tonsil be inculturated as harboring the focus of infection in cases of chorea.

3. There should be a close working association between all the branches of medical specialization if the best service is to be given in conditions dependent on focal infections.

4. All areas of infection should be cleaned up after removal of the tonsils, and appropriate after-treatment vigorously pursued.

SOME UNUSUAL DISEASE CONDITIONS APPARENTLY CURED BY TONSILLECTOMY

REMARKS ON POSTOPERATIVE TREATMENT. PRELIMINARY REPORT

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That the faucial tonsils are frequently the source of serious systemic infection and may be the very fountain head of metastases is now so well known that I shall proceed on the assumption that this is a conceded fact. Yet, in many of my cases, so innocent was the appearance of the tonsils, and so few were the subjective symptoms elicited, that I should have declined to remove them had not a competent internist already eliminated all other likely sources of infection. It is most important that this step should be first taken in all obscure cases of arthritis, Bright's disease, retinitis, and other states where a distant focus might be supplying the toxin causative of the disorder under investigation. There are, of course, perfectly frank cases, in which the tonsils are obviously of the toxic type and need no confirmation of their guilt in causing the disease. A laryngologist may feel confident that a given appearance in a tonsil indicates its possible responsibility for a disease condition, but unless an internist of experience and judgment has first eliminated other possible causes, he would be showing poor judgment, if not actual reprehensible carelessness, in removing the tonsils. Shambaugh¹, whose wide experience and ripe judgment command his opinion to our ready acceptance, says that "the throat specialist ought not to assume the responsibility of passing on these questions. They are problems that fall more properly in the work of the internist, and often constitute one of the most difficult problems for him to solve."

Apical tooth abscesses have been shown capable of causing all the metastatic affections formerly attributed to diseased tonsils, and frequently both conditions are found in the same case. Where this coincidence of toxicity occurs, it is my belief that a careful history will reveal the fact in most instances that the teeth were the original source of infection, and, in fact, caused the tonsillar degeneration.

1. Shambaugh, G. E.: *Ann. Ophth. Rhin. and Otol.*, March, 1917, p. 137.

In view of the great sorrow to the patient, and the enormous economic loss to society, by blindness, or even marked loss of vision, perhaps the most brilliant result of tonsil removal is that secured in clearing up the effusion in choroiditis and chorioretinitis of infectious origin. These cases so often proceed to blindness or serious loss of vision, and with such rapidity, that any treatment which arrests the process, and even clears it up, cannot be overestimated. Thus, in some forty odd cases which were referred to Drs. A. Y. P. Garnett and W. C. Moore for physical examination by various ophthalmologists, the tonsils were found responsible in 35 per cent., teeth 60 per cent., sinuses 5 per cent. It is well to invite attention to the fact that these cases were first seen by the oculist, then by the internist, and finally by the dentist and laryngologist; treatment was given as indicated by the findings of this group of examiners, with the eye specialist in control. So slight was the manifestation of tonsillar disease in many of these cases that I was reluctant to operate until I had had a consultation with the internist; yet every case showed definite disease areas beneath the plica, after dissection.

REPORT OF CASES

CASES 1, 2, and 3.—*Chorioretinitis*. The following three cases illustrate three different ways in which a diseased tonsil may create an inflammatory effusion in the eye.

(A). S. T. R., man, aged 24, had good general health. Dr. R. S. Lamb reported a mild but definite chorioretinitis with exudate, and suggested an examination of the tonsils and teeth. The teeth were negative. The tonsils were removed in part five years before under local anesthesia, but I found a large piece in one fossa, and a small segment completely invested in the plica on the other side. Under ether both remnants were removed with much difficulty, and some pus and caseous material found beneath both capsules. Absorption of the toxin had been inevitable, as the tonsil tissue was absolutely hidden by the scar and mucous surfaces. Cure was complete in six weeks. This case illustrates the futility, and even harm, of an incomplete operation, aside from any ethical considerations.

(B). H. L. W., woman, aged 27, had good general health but was rather nervous. Eye report by Dr. J. W. Burke. Seven years before the patient had disseminated chorioretinitis in right eye, but no trouble was ever found in left, though she had had a "black spot before left eye"; exudative chorioretinitis found with great deal of exudate; no syphilis. Wassermann test made two years before. After thorough examination by internist, I found her tonsils very suspicious and removed them. The next day the exudate was enormously increased which was, of course, entirely confirmatory of the part the tonsils had been playing.

In a month the eye was normal. This patient had been examined by a man of international reputation, who handed her over to a rhinologist, who also did eye work; hence it is all the more astonishing that this specialist did not see the tonsillar disease (pus was secured by probing), but even did a submucous resection on her septum. While the disease in the tonsil was not very obvious, yet the diagnosis by exclusion was so compelling—and a deviation of the septum never yet caused a metastatic infection in the absence of sinus disease—that the tonsils should have been scrutinized with the greatest care. Had they been so examined their responsibility would have been manifest. This case illustrates the necessity for judgment and power of observation in an examination.

(C). G. D., woman, aged 31, had good general health but was nervous at times. Eye report by Dr. W. H. Wilmer. Headache for many years after reading, or exposure to glare, unable to read for more than ten minutes without suffering afterward well into the next day. At one time did no close eye work for a year. Central chorioretinitis. Physical examination negative; tonsils found diseased only on being drawn out of fossae and probed. Wassermann reaction weakly positive. Removal of tonsils showed extensive follicular disease, and resulted in eyes being restored to perfect functional health in three weeks. She had been refracted many times in efforts to relieve the pain and weakness of the eyes, but never had had her tonsils or teeth called into question. This case shows merely ignorance on the part of the original oculist. The eyes in all these cases received appropriate local treatment, such as dionin, pilocarpin, and potassium iodid, which assisted in hastening absorption, but did not remove the cause of the effusion.

CASE 4.—*Chronic Indicanuria*. H. H. H., man, aged 37, fair general health; more or less trouble with the tonsils since childhood, but nothing serious. Typhoid fever eight years before, serious case. Indicanuria for ten years without interval; constant efforts at eradication without success. For past one and one-half years subnormal temperature, ranging from 95 F. morning to 97 F. evening. During this period anemic, dispirited and listless. Diagnosis of adrenal insufficiency made (and very likely true) by competent internist; pulse 60, systolic pressure 95. Consulted me for relief of fulness in ears and slight deafness, subsequently found to be due to congestion of the mouth of the eustachian tube, an extension of a chronic inflammation of the tonsils. These glands were seen to be buried, moderately enlarged and extending very high into the supratonsillar fossa. Catheterization always relieved all symptoms, but only for a few days at a time. Removal of the tonsils, a perfectly obvious necessity, revealed deep disease and considerable pus, and resulted in permanent relief of the symptoms for which the operation was done as well as of all those previously thought due to the adrenal trouble. The chronic presence of indican was not known to me, but, on my remarking on the astounding change in his whole

attitude to his work and the marked physical improvement, he mentioned that the test made that day (two weeks after operation) showed the absence of indican from the urine for the first time in ten years; and it has continued absent after some ten months. The urinalysis before operation showed it in great amount.

CASES 5 and 6.—*Multiple arthritis and Bright's disease (hyaline casts, albumin, high blood pressure and headache)*. Two cases.

(A). M. W. T., woman, aged 43, fair general health; practically all joints involved, pain intense and swelling great, albumin and hyaline casts for 10 years; severe migraine for 5 years, during past two years rarely absent for more than three or four days at a time. Eyes showed chronic disseminated chorioretinitis. Typhoid fever twenty years ago. Roentgenogram of teeth showed numerous suspicious teeth, which had been always well cared for. Tonsils deeply buried, but had never given any serious trouble. However, at operation they were found sufficiently toxic to account for many if not all of her symptoms. The teeth were extracted but showed no apical abscesses, though much caries in the upper portion. However, all teeth had been regularly filled and treated for years by competent dentists; so I believe they exercised little if any effect on the general condition of the patient. For the first six months after operation the patient had not one of the objective or subjective symptoms, and has had none of them return save a few headaches in the past two years.

(B). M. B. H., woman, aged 45, general health poor, very nervous. Recurrent attacks of mild sore throat and severe arthritis in all large and many small joints, with great swelling and temperature up to 101 F. Teeth defective but well cared for. Hyaline casts, albumin in urine for several years (present day of operation); deposits in retina from old disseminated chorioretinitis; headache often. Tonsils flat, but showing numerous crypts and some pus on probing. At operation one tonsil showed much detritus, and sharp attack of arthritis was caused in 24 hours. Culture of tonsils gave pure streptococcus, from which vaccine was made and given for some ten weeks. Convalescence slow and pain left very gradually, but all symptoms are still absent after year and a half. The casts and albumin disappeared in two days and have not returned. Headache is still very rare, though stiffness without swelling or pain in joints occasionally manifests itself.

CASE 7.—*Darier-Roussy Sarcoïd*. This term is the only one under which the following case can be classified, in which Dr. H. H. Hazen assured me the lesions are absolutely identical. He referred the case after an examination of the patient from top to toe, including the tonsils which he said offered the only possible clue to an etiology.

F. C., man, aged 28, complained of painful nodules on arms

and legs, situated near large vessels and adjacent to bone, varying in diameter from one-half to 1 inch, deeply situated in the corium. Sections showed a panphlebitis and panarteritis with a surrounding infiltrate of epithelioid tissue (report of Dr. Hazen). In the seven years of his trouble the patient had consulted the best internists and dermatologists in the country, but had never had any relief or any real diagnosis till Dr. Hazen took hold of his case. Repeated Wassermann tests were consistently negative, and I found his tonsils certainly toxic, though not enlarged. Neither Dr. Hazen nor I had any very roseate prognosis for the result of the tonsil removal, but, as they were diseased and had given him some slight discomfort in the past, and as his suffering from the nodules was constant and severe, the operation was a conservative and wholly justifiable measure, and such it proved. As usual, the tonsils were found much more diseased than they outwardly suggested, having many minute pockets of pus throughout their substance. In two months all the nodules disappeared and did not return till eight months later, when a gingivitis and pyorrhea alveolaris caused a slight recurrence. This relapse is now under control, and but strengthens the theory of the tonsillar origin, one of the focal variety. The return is thus obviously due to the development of a new focus, and this too is being proved by the disappearance under treatment of the nodules, directed at the gum and alveolar process disease.

COMMENT.

Since the almost universal adoption of total removal of the tonsils as the preferred tonsil operation, there has accumulated much evidence that the cosmetic and functional results have been often disappointing and even disastrous in their local effect. In a few cases this is unavoidable, but often the cause lies in failure to treat the operative wound persistently until healing has taken place. Where this neglect has occurred the fossae are invaded with quick growing granulations and their subsequent organization into hard, irritating masses causes discomfort and interference with the vocal function of the tonsillar pillars. The deforming contractures which are seen at times are due to a totally different cause, that is, failure to conserve the pillars at operation. After experimenting somewhat I have adopted a definite routine of postoperative treatment of the tonsillar fossae which has given me encouragement despite the occasional faulty results. After leaving the hospital the patient reports daily at the office and there the fossae are first sprayed freely with peroxide, the froth is then sprayed away with any bland alkaline wash and lastly the now thoroughly cleansed cavities are swabbed with any form of iodine and glycerin one may prefer. For the first two applications a spray is to be

preferred to a swab as it penetrates much better, removes the detritus more thoroughly and is much less, if at all, painful. The pain which is always present, and the edema which is nearly always present in some degree, can both be much relieved and at times abolished by hourly normal salt solution irrigation of the fauces at a temperature of about 110 F., beginning three hours after operation and kept up as long as the patient is in the hospital. This treatment possesses the further advantage of materially hastening the healing process and removing the thick tenacious mucus, whose forcible expulsion on the part of the patient is so painful, and whose retention in the mouth so provocative of gagging, itself distressing and apt to lead to hemorrhage. Nasal irrigation is likewise helpful in that it removes the clots and reestablishes nasal respiration which relieves the discomfort of mouth breathing, one of the lesser causes of the patient's suffering. It should not be done more than twice after operation, however. The application, while the patient is still in the hospital, of various iodine solutions does not appeal to me, as the fibrinous covering which nature at once throws over the denuded surfaces is distinctly protective and even germicidal, and for this reason should not be disturbed for two or three days, at the end of which time it will have lost these properties and become a slough. This, of course, is no longer protective but an obstruction to the escape of noxious material. My objection to the early application is not only theoretical, as set forth, but eminently practical, as I have tried it faithfully and found it in no wise superior to the technic I have devised myself, one doubtless many others follow without my knowledge. Moreover, the pain it causes is always intense, and sometimes excruciating, which condition causes the reflection that we pay too little attention to the patient's feelings in many of our manipulations. When a patient says a certain procedure hurts, the remark is often not heeded, unless we reply that it is unavoidable; whereas it is generally unnecessary. Grasping the tongue in laryngoscopy, separating the alae of the nose or even depressing the tongue, that simplest of procedures, are all painful or free of pain in proportion to the dexterity or consideration of the examiner. Roughness of manipulation bears no relation to thoroughness, nor does gentleness prevent a searching inspection. Inasmuch as a patient will submit to a more thorough examination and treatment at the hands of an attendant who evinces some desire to avoid unnecessary pain, I submit that this consideration is not purely academic but eminently practical.

All patients are not aware that the Stoics regarded pain as one of the lesser pleasures, and probably would not subscribe to the doctrine if they did.

In all the cases reported the frequency with which the tonsils, outwardly but little diseased, have been shown as harboring serious toxicity, the source of metastatic disease, is striking. However, I wish to state unequivocally that I am reporting seeming cures and not recommending tonsillectomy for the relief of any of the conditions discussed herein, not excepting arthritis, unless the patient has already been thoroughly examined by a competent internist who has excluded other sources of infection; even so, the tonsils must appear at least abnormal, if not actually toxic.

The abuse of the tonsil operation has grown to such an alarming extent since the promulgation of the focal infection theory that I do not wish to appear to lend my support to any increase in the list of diseases which justify tonsil removal. It is my creed that no child's tonsils should be removed unless it is possible to demonstrate a definite connection between those tonsils and some pathologic condition in that child. On the other hand, given an adult suffering from a disorder commonly associated with tonsillar disease, it is justifiable to remove these tonsils if there is a reasonable suspicion attached to them. I have seen four patients die of endocarditis (of proved tonsillar origin) from neglect of this precaution, though none of them had anything more severe when I saw them than a history of repeated attacks of tonsillitis associated with slight pains in the joints. The tonsils were plainly diseased though not much enlarged, except in one case. The deaths occurred, one in four years after my advice, the other three within two years, and none of them had any heart lesion or functional irregularity when seen by me. The heart in each instance was examined by a diagnostician of unquestionable ability; so I know the lesions which resulted in death were not present when I suggested operation, and hence I feel sure that the deaths were avoidable. One of the cases was especially sad, in that she was quite willing to submit to operation but her family physician dissuaded her. It is hardly to be questioned that her death lies at his door.

TONSILLAR INFECTIONS AS A SOURCE OF SYSTEMIC DISEASE

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NEW YORK

Many contributions to the literature within the last few years, as well as much research work, have proven definitely that infections of the tonsils are frequently the cause of such systemic conditions as infectious arthritis, acute rheumatic fever, nephritis, endocarditis, myocarditis, anemia, cervical adenitis, goiter, chronic coughs and colds and a large group of nervous disorders commonly classified as neurasthenia.

The organisms found in the tonsils in such conditions are the usual pathogenic varieties, such as the *Streptococcus viridans*, *Streptococcus hemolyticus*, *Staphylococcus albus*, pneumococcus, tubercle bacillus, several varieties of gram-negative diplococci, the *Bacillus pyocyaneus* and colon bacillus. In the author's experience the *S. viridans*, *S. hemolyticus* and a gram-negative diplococcus, have been most frequently encountered in the systemic conditions. Occasionally secondary infections, such as syphilis and sarcoma, occur in the tonsils. The *S. viridans* shows especial affinity for the heart valves, and the *S. hemolyticus* for the joints. Focal infection is the most important subject before the medical profession today. A focus of infection may be located anywhere in the body, but certain structures are more frequently affected than others. An ideal site for a focus of infection is in a structure which has a recess or pocket that does not drain freely. Most infections are harmless as long as free drainage is maintained. When, however, the drainage from an infection is shut off, toxins are produced and absorption takes place. Experience has taught, therefore, that the tonsils, teeth, nasal accessory sinuses, mastoid cells, pulmonary alveoli and bronchi, gallbladder and ducts, appendix, uterus and fallopian tubes, prostate and seminal vesicles, pelvis of kidney and urethra frequently harbor a focus of infection. The tonsils, on account of their location in the oral cavity and their histologic structure, are the most frequent site for these infections. Absorption of toxins from intestinal stasis is capable of producing the same conditions.

The location of the tonsil makes it especially susceptible to infection. The faucial tonsils are situated on either side of the oropharynx, and are bounded in front and behind by the faucial pillars, and externally by a capsule. Their inner surface is covered with stratified epithelium. Each tonsil contains from eight to sixteen crypts or lacunae, branching out and extending nearly to the capsule. These lacunae or crypts contain broken down cells and débris, and often lodge particles of food. If the openings of these lacunae or crypts become closed the matter is retained and becomes fetid and laden with bacteria. The surrounding tonsillar tissue becomes inflamed. The glands at the angle of the jaw then become enlarged, and furnish evidence of the absorption of septic material from the chronically inflamed tonsil. The fetid masses are more frequently found in the buried tonsils, the character of which is likely to escape notice. In such tonsils it is worthy of note that the margin of the faucial pillars is more congested than the rest of the mucosa. When this congestion is evident the tonsils should be carefully examined. In tonsillar infections the other lymphoid tissue in this region, that is, the adenoid and the lymphoid tissue at the base of the tongue, are frequently infected. They should not be overlooked in searching for a focus of infection.

Coakley in a recent paper quotes the following experimental work done by Dr. S. Flexner at the Rockefeller Institute. The tonsils were removed from patients under aseptic precautions and placed in sterile containers without being handled, and delivered to the Rockefeller Institute. This was done in connection with animal inoculations from tonsils of patients suffering from poliomyelitis.

"Cultures of streptococci were injected into rabbits intravenously, the cultures in the first one to three generations on artificial culture media being employed. Altogether the number of rabbits injected from cultures made from tonsils was 154, of which 101 succumbed to the inoculation. Of these animals eight developed paralysis of one or more limbs; twenty-nine developed meningitis, and about two-thirds of the rest developed arthritis. About one-half of the cultures came from the tonsils of poliomyelitis patients, and the other half from patients who were nonsufferers from poliomyelitis. The distribution of the lesions in the two series was almost identical. The most frequent effects were infections of the joints. Endocarditis, pericarditis, abscess in the muscles, and abscess in the kidney developed in a few animals as a result of the inoculations."

Such work is exceedingly valuable corroboration of the clin-

ical evidence that such lesions frequently follow acute infections of the tonsils.

Dr. G. B. Wood in an experimental study has proved that the streptococcus and other varieties of organisms can enter the blood through the medium of the tonsils and produce infection in the joints, the heart or the kidneys. He has also found that the tonsil in the hog is more readily infected by the anthrax bacillus than any other portion of the buccal mucous membrane.

In 1910, H. W. Loeb cited four cases before the American Laryngological Association and said: "Acute nephritis results from acute tonsillitis far oftener than is generally supposed."

In chronic arthritis the focus of infection has usually been found in the head, and most frequently has been found to be a chronic infection of the tonsil.

Davis obtained pure cultures of *S. viridans* in the crypts of 40 per cent. of the tonsils removed from patients with endocarditis which was supposedly of tonsillar origin.

Osler says that, "tonsils are probably the port of entry for the microorganisms of the not infrequent cases of endocarditis without recognizable cause. In acute endocarditis of tonsillar origin, if the patient can survive the anesthetic it is better to enucleate the tonsils than to permit their continued transference of toxins."

Harbitz, in 1905, made a careful study of the glandular systems in a large number of clinically tubercular persons and others, with the object of determining the port of entry in those with tubercular lesions of lungs, glands, bones and meninges. In many instances he demonstrated by animal inoculation and by cultural methods tubercle bacilli in the tonsils, adenoids and cervical glands that grossly and microscopically showed no tubercular lesions. It is, therefore, probable that the tubercle bacillus may not only pass through normal mucous membrane without causing lesion, but may remain latent in histologic normal lymph glands for months or years.

Dowd finds in tuberculosis of the glands of the neck the oldest lesion in the so-called tonsillar gland at the angle of the jaw in 86 per cent. of his cases. Many of his patients give a history of frequent attacks of tonsillitis or pharyngitis.

In searching for a focus of infection in systemic conditions, a careful history of the patient's illness is important. We believe that the absorption of chemical toxins from these organisms in the tonsils is capable of producing serious lesions in the nervous system, in the heart, kidneys, joints, muscles, glands,

blood and respiratory tract. These symptoms or sequelae may be so serious that life itself is endangered. All of these complications have been observed in our clinical experience. Improvement and cure have followed the removal of these infections in numerous patients who had previously resorted to every known means of treatment, whether it was an endocarditis, myocarditis, nephritis or arthritis.

By this it is not meant that every patient with these affections in which these organisms have been found in the tonsils has been cured, but that we have a record of at least one such case in each type in which the eradication of the focal infection in the tonsil has stopped all symptoms.

CLINICAL HISTORY

The typical clinical history of such cases is as follows:

The patient had a tonsillitis of greater or less severity, and sometimes even no discomfort was felt in the tonsils but only an infection in the nose and throat was noticed. Within a few days or a few weeks some other symptoms developed. The tonsillitis or infection of the upper respiratory tract may have been so slight that it was entirely forgotten by the time the complication developed. If the complication was arthritis, or the so-called rheumatism, it usually developed shortly after the initial tonsillitis. As a rule the more severe the attack of tonsillitis, the sooner have the complications manifested themselves.

Our observation has been that if the tonsillitis is severe the complications generally ensue quickly; if mild they develop later in from one to eight weeks. In our own patients who have been seen during the initial attack, and treated according to the plan to be described below, no complications have developed after the treatment was commenced. The complications usually have developed without a chill at their outset.

PROGNOSIS

The prognosis of acute tonsillitis in which these organisms are found, varies greatly with the treatment. All of our patients who have been treated with the autogenous vaccines have been relieved of all symptoms and no complications or sequelae, such as rheumatism and arthritis have developed. Patients treated in this manner have made unusually rapid recoveries from tonsillitis. Some of the acute cases were complicated by arthritis and nephritis, but these also have been cleared up promptly by the treatment.

On the other hand we have seen many patients in consultation long after the initial tonsillitis, who consulted us because of some obstinate condition such as arthritis or some cardiac condition, and these organisms have been found in the tonsils as long as five years after the initial attack of tonsillitis. Many such patients have been relieved by the administration of autogenous vaccines and the enucleation of the tonsils.

We are, therefore, led to believe that some of these complications might have been averted by the prompt administration of an autogenous vaccine during the acute tonsillitis. The pain and swelling in arthritis or rheumatic joints, in a considerable number of patients, of from one to five years duration have been relieved by this treatment. In some cases, in which great changes or bony ankylosis and exostosis have taken place in the joints, the treatment has stopped all evidence of activity as pain, redness and swelling of the soft tissues, leaving only the deformity. In a group of cases in which in addition to the joint symptoms there were cardiac and renal changes, great improvement in these conditions has taken place.

The symptoms, complications and sequelae of these infections can best be described by reciting cases which illustrate types. These will follow after a few words in a general way on the treatment.

TREATMENT

This consists in eliminating the infection. The infection that causes the trouble is situated in the crypts of the tonsils. The culture is taken from the crypt of the tonsils. The surface of the tonsil is swabbed with 95 per cent. alcohol to get rid of mouth contamination. The sterile platinum loop is then immediately passed into a deep crypt of the tonsil and inoculated on a culture plate. The plate is incubated for twenty-four to thirty-six hours at body temperature. The organisms are then identified and an autogenous vaccine prepared. We prepare our vaccine so that 1 c.c. represents 200 million of the organisms. This vaccine is then administered to the patient three times a week until all bacterial activity has ceased, and then the tonsils are enucleated unless there is some contraindication for their removal. The teeth are frequently found to be infected, and if so the patient is referred to a competent dentist for appropriate treatment.

We believe that the administration of the autogenous vaccine not only stops the activity of the organism by overcoming the effect of the absorption of its toxins, but that it renders tonsil-

lectomy a safer and more satisfactory operation. Frequently after tonsillectomy there is a low grade sepsis which is not only annoying to the surgeon but also endangers the patient's life. For instance, we have seen several patients whose tonsils were enucleated for the relief of rheumatic pains immediately have a subacute exacerbation of the attack which continued indefinitely.

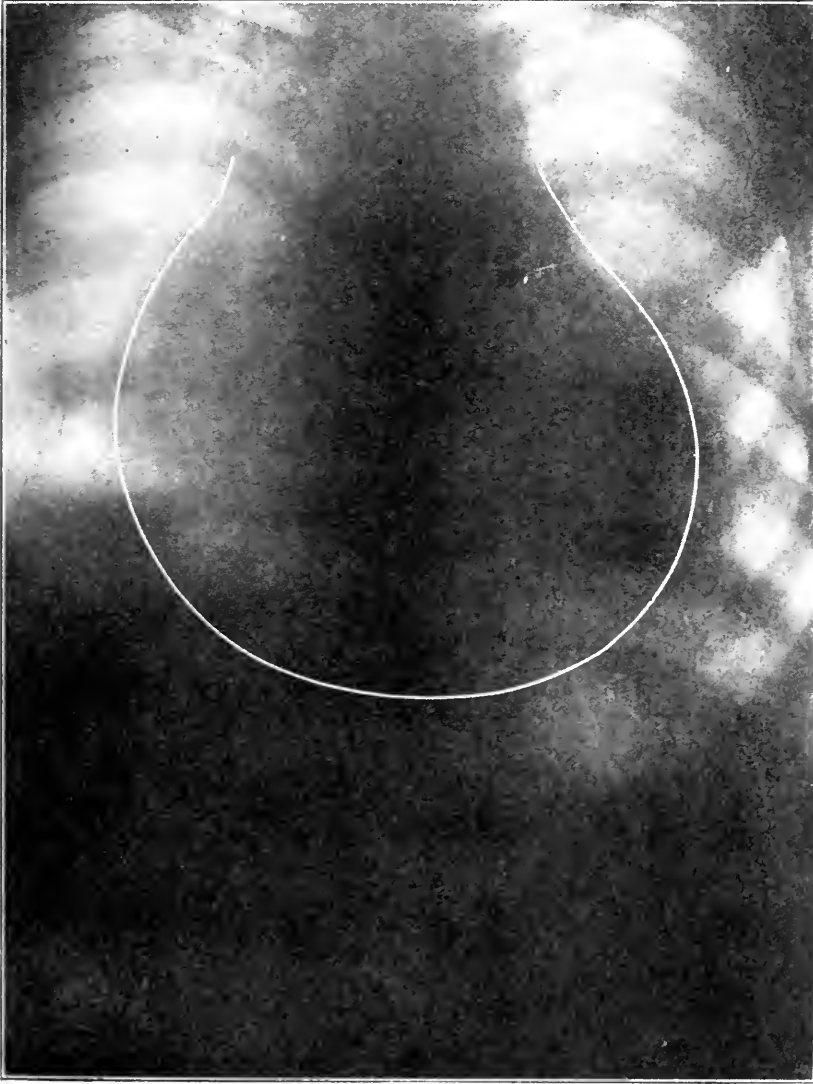


Fig. 1.—Cardiac radiograph of J. S., case I, showing enormous hypertrophy of heart at the time treatment was begun with autogenous vaccine made from the crypts of his tonsils. (Courtesy Medical Clinics of N. A.)

and the disease for the relief of which the tonsils were removed was not helped. Our experience with the autogenous vaccine treatment of these patients leads us to believe that much of this trouble can be averted by its use as a preliminary step to the operation.

The case reports which illustrate what has been said in the foregoing will be brief. Their complete histories can be found either in our hospital or private records.

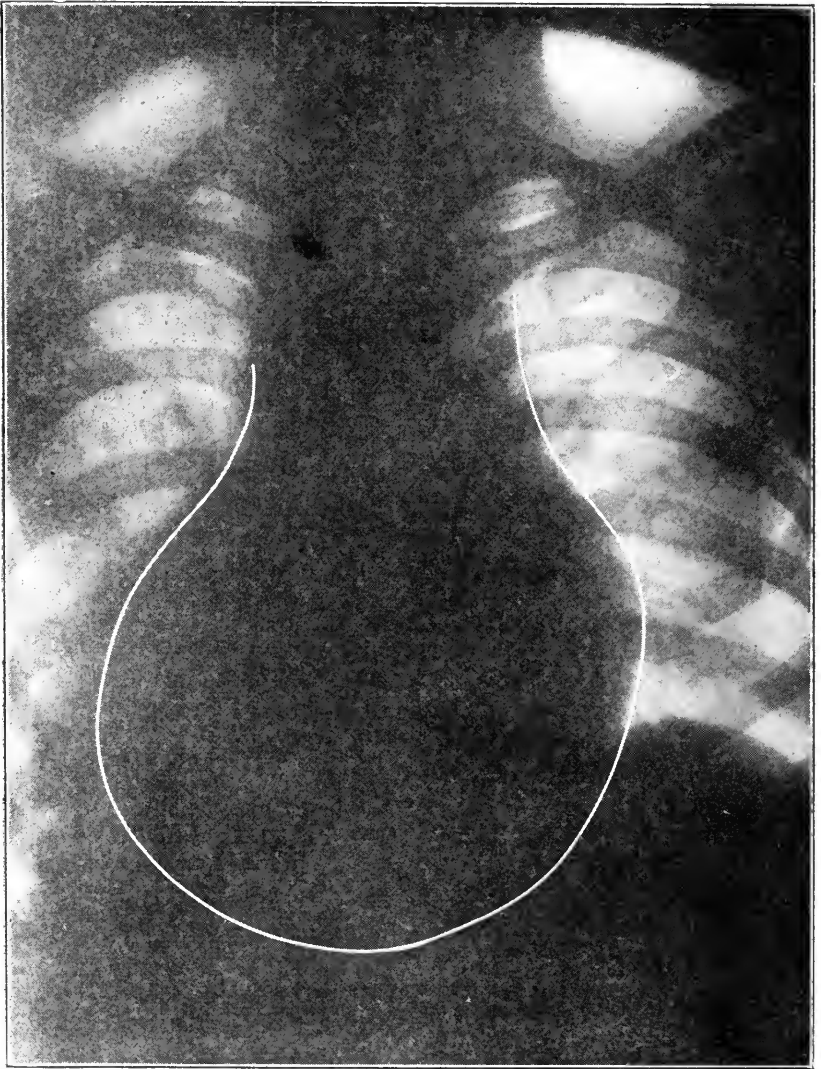


Fig. 2.—J. S., same heart as Fig. 1, radiographed about three weeks later, when the patient was greatly improved. (Courtesy Med. Clinics of N. A.)

REPORT OF CASES

Acute endocarditis, complicated by pericarditis with effusion, due to Staphylococcus albus infection of tonsils. Marked improvement following the eradication of this infection.

CASE 1.—J. S., man, aged 27, was admitted to Gouverneur Hospital, March 23, 1917. The diagnosis of acute endocarditis, complicated by pericarditis with effusion was made. His temperature on admission was 104 F. He was dyspneic, with cyanosis of the lips and pallor of face. He had all the signs of being acutely ill. Tonsils were congested and painful.

Summary of Examination on Admission.—Hypertrophied heart, mitral regurgitation, painful joints, cyanosis of lips and extremities, dicrotic pulse, inflamed tonsils, septic temperature.

History.—This man gave a history of troublesome tonsillitis and rheumatism during his past life. The tonsils caused him almost constant trouble during childhood. When he came to the hospital his heart was enormously enlarged and in a poor state of decompensation. The roentgenogram revealed a heart shadow which almost filled up the whole lower thorax and abdomen.

Treatment.—For four weeks he was treated in the usual way in the hospital. His condition became worse. About April 20, we were asked to examine his tonsils. A culture made from the crypts showed a pure culture *Staphylococcus albus*. At that time, he was so sick that all of us expected his death at any time. We had an autogenous vaccine prepared from his culture and gave him 200 million by hypodermic injection every second day.

Much to the surprise of all of us who had seen him, his improvement was rapid and continuous under the administration of the vaccine.

The following note on his condition was recorded on April 30 after he had received about five injections of the autogenous vaccine.

"Pericardial effusion was being absorbed. Area of cardiac dulness very much smaller. No friction. No cardiac decompensation."

Results.—He left Gouverneur Hospital May 20. His general condition as well as his cardiac condition was greatly improved. All of us who saw him when he was so desperately ill, marvelled at his recovery. Our opinion was that his tonsils should be enucleated, but he was satisfied with his improvement and refused to stay longer in the hospital.

We might add a word in general on the treatment of such patients. We found a focus of infection in his tonsils, the staphylococcus albus and the autogenous vaccines apparently cleared up his condition very rapidly. It is difficult to absolutely prove medical facts, but it appears that the vaccines saved the man's life. He was gradually getting worse under ordinary

treatment—digitalis, sodium salicylate and bicarbonate of soda, but as soon as he received vaccines his improvement began.

We who have seen a considerable number of such recoveries are therefore convinced that the vaccines are efficient in suitable cases.

(This case is reported more fully in the New York Number (November, 1917) of The Medical Clinics of North America published by W. B. Saunders Co.)

CONTRAINDICATIONS

We have encountered some opposition both by physicians and patients to the removal of tonsils in patients with cardiac lesions.

It has been our privilege to operate on a score or more of patients with various cardiac lesions and without exception, such patients have withstood the operation well, and usually the heart lesion has improved after the tonsils were removed. These patients were operated on because it was believed that the focus was in the tonsil. The question of tonsillectomy for such patients should, therefore, be carefully considered.

ANESTHESIA

The question of anesthesia in these patients should also be carefully weighed. We have operated on some under ether and others under local anesthesia, considering carefully what is best for the particular individual. It seems to us that just

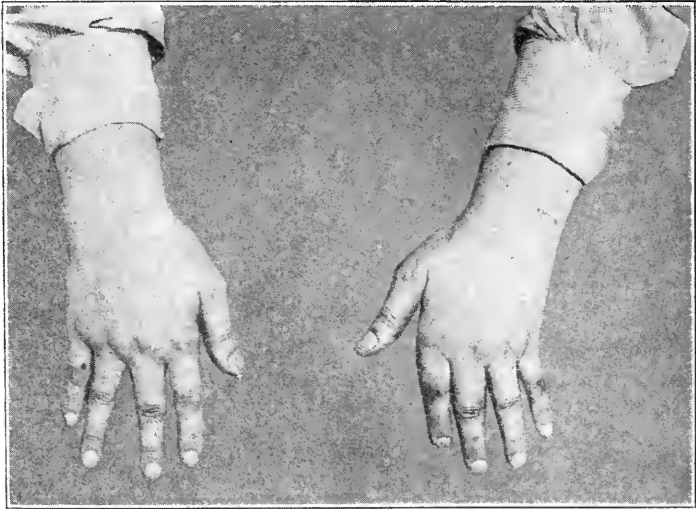


Fig. 3.—D. B. Infectious arthritis of about ten years standing. This photograph was taken July 31, 1917, when treatment was commenced. (Courtesy Med. Clinics of N. A.)

as a focus of infection is searched for in arthritis, so also should a focus of infection be assiduously looked for in lesion of the heart, such as myocarditis and endocarditis. We have frequently seen cardiac murmurs disappear and the heart become normal in function after the focus of infection has been removed.

Cervical adenitis. We have had a number of cases of involvement of the submaxillary glands due to a focus of infection in the tonsils. The organisms found most frequently in our experience have been the *Streptococcus viridans* and a gram-negative diplococcus. These two organisms have usually been found associated in each case.

CASE 2.—M. P., a boy aged 16, was first seen when the glands at the angle of his left jaw were matted together in a

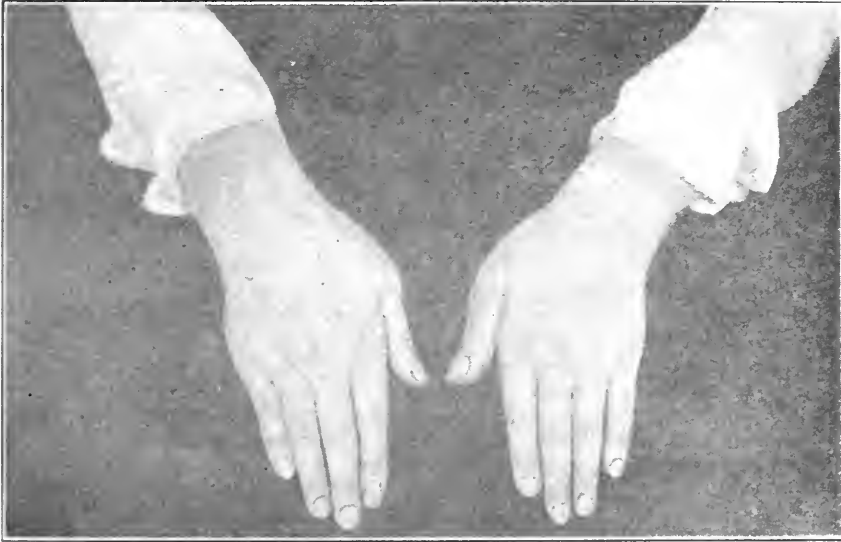


Fig. 4.—D. B. Same hand as in Fig. 3. This photograph was taken about five weeks later, following treatment with autogenous vaccine made from the crypts of tonsils. Her infection was a gram-negative diplococcus. (Courtesy Med. Clinics of N. A.)

mass as large as a golf ball. They were hard and we suspected a malignancy of the tonsil. The tissue removed from the tonsil was negative for malignancy but cultures made from the crypts showed *S. viridans* and a gram-negative diplococcus. His tonsils had been operated on once or twice previously but the greater part of the left tonsil was still in situ.

He received autogenous vaccines and the glandular mass rapidly disappeared. He has had no recurrence during the three years that have elapsed since he was treated.

CASE 3.—B. S., a young college man, aged 21, was referred in November, 1916, on account of enlarged submaxillary glands.

There were two submaxillary glands on his right side, one was the size of a hen's egg and the other about the size of a pigeon's egg. His tonsils were enlarged but there was no history of tonsillitis. Cultures from the crypts showed *S. viridans* and a gram-negative diplococcus.

In two days after the first injection of autogenous vaccine, there was a noticeable decrease in the size of the glands. After five injections the glands were palpable but not visibly enlarged. His tonsils were then enucleated and he made a rapid and uneventful recovery. No recurrence during the one year that has elapsed since his first visit.

There are many cases of chronic infectious arthritis that might be reported from our records showing very happy results.

CONCLUSIONS

1. Focal infections especially those of the mouth, tonsils and teeth and alimentary tract, to use the words of Professor Osborne of Yale, are "no fads" but a reality to be looked for and eradicated if we are to cure our patient.

2. Autogenous vaccines produce remarkable results quickly in suitable cases.

3. These foci of infections should be searched for, in all patients but especially those afflicted with cardiac lesions, arthritis, cervical adenitis, goiter and lesions of the nervous system too often passed up as neurasthenia.

DISCUSSION

DR. ALBERT H. ANDREWS, Chicago: To some of us who have been in the work a long time the question naturally arises where have we arrived, and how did we get there? I remember a few years ago reading a number of papers before general societies on the subject of the constitutional effect of diseased tonsils. I was not the leader in that respect. Dr. Pynchon was the man to take the lead. But I got the credit for being very advanced in my ideas, but had I read such a paper then, as I heard this morning, I would have been considered insane. The credit for popularizing this subject belongs to a well-known and able practitioner, Dr. Frank Billings. I remember also attending a meeting of the American Medical Association in Saratoga Springs and sitting by the side of one of our very able men who is with us this morning, Dr. Emil Mayer. Some one read a paper on the subject of the removal of tonsils and I tried to make three points. He told how quickly he could remove tonsils. I protested against tonsil snatching, I protested against decapitation of the tonsil versus enucleation, and I protested against holding children in removing their tonsils. So long as we continued that we should not be surprised at the number of anarchists increasing in this country, especially in the young. There is no question that diseased tonsils produced constitutional disturbances in distant parts. But just when and where and how it is done is a question. It is also a question as to which tonsil should be removed and which should be let alone. Some two or three years ago I took up work in an undergraduate school and one of the examination questions I asked was to

name the deleterious effects of diseased tonsils. The students had been under the care of the general medicine man, and the surgeon, and the genito-urinary specialist, and the neurologist, and incidentally they had been under my instruction, and the list the students gave of the deleterious effects of diseased tonsils would do credit to a modern patent medicine advertisement. If some one will give us a clear rule for determining which of the questionable tonsils should be removed and which left alone he will confer a very great favor on humanity and help out the medical profession. I also asked the question: Give the indications for removal of the tonsils? One student summed the whole question thus: The presence of a tonsil indicates an operation.

DR. EMIL MAYER, New York City: Usually the question of tonsils and what to do for them is an important one. In the first place, we ought to be prepared to agree with each other. It is not advisable for one to say they should come out and the others think they should not. Therefore, systematize our examinations. The ordinary examination is made by putting a tongue depressor in the mouth, observing the tonsil and then expressing an opinion as to that tonsil. Consider everything in connection with the throat as well. In the first place, if that tonsil is to be examined, examine it properly. It should not only be transilluminated, but, as suggested by Dr. King, put into a platinum loop and the crypts examined. I consider Dr. King's suggestion seriously. I have followed his work for some years, and have upheld him in his contentions with genuine pleasure. Next, take your compressed air and blow out the crypts. You will be surprised to see many and many a tonsil give out caseous pus. Then, another thing in the throat is the uvula. How many take the trouble to tease it down to see whether there is a little papillomatous tip? So the neighboring organs should be looked at. Get your postnasal mirror and examine the uvula posteriorly. Diagnosticians are woefully ignorant as to throat conditions which has made for much unhappiness. One woman was given a diagnosis of that abomination of medicine—hysteria. I don't remember when I have seen more cheesy tonsils than that woman had. Now it becomes all important for our operations to be thorough. I consider very favorable the remark originally made, I think, by Dr. Marcus of Chicago: Paint the enucleated surface with iodine before taking the patient from the table. Now we have one thing to pay attention to particularly and that is, we must not say hard and fast that a patient must be operated on. There are patients who do not wish an operation for reasons, or there may be a case in which there is a history of bleeding. I would say that those latter are the cases in which deep cauterization and thorough exenteration of the tissue between the crypts is of value. As to the post-operative treatment of these cases I am quite sure that most of us do neglect them as has been suggested by Dr. Dabney. We are happy to know that the patients have left the hospital, and we let them go for several days before we hear if they are well. Dr. Andrews recalled to my mind that the burden of all our song must be that we must try to agree which tonsil is to be removed and which tonsil left alone.

DR. CHARLES S. MEANS, Columbus, Ohio: I wish to thank Dr. Wright for his timely and opportune paper. I am glad Dr. Wright has put his disapproval on partial removal of the tonsils in which I fully concur, yet I do not quite agree with his conservatism in the prognosis. While it is not possible to tell that there is a positive cure for tonsillar infections, yet my experience has been that I have given a great many people relief by removing the tonsils when there was not much to prove that

the tonsils were the infecting foci, yet it proved to be such. If relief was not given I feel satisfied no harm was done.

I have removed tonsils from all ages ranging from 4 months to 72 years. I removed them from a woman of 72 with no untoward symptoms whatever. So we have no reason to limit ourselves in age, if operation is necessary for the desired relief.

I have had two experiences in the last few weeks with children who had their tonsils "split" in years gone by. These children had acute tonsillitis followed by arthritis and endocarditis. Each had been in bed over a year not being able to be up at all on account of a leaking heart. They have both recovered following complete enucleation of the tonsils and are now in school.

I disagree with the essayist that dissatisfied patients do not return. My experience has been they return more often than the satisfied ones much to my discomfort.

I fully agree with the procedure of total enucleation. Some people will say that the superior lobe only should be removed, but I have never had any satisfactory experience in removing by piecemeal. I have always been sorry when I attempted this operation. Of course, we have infected teeth and sinuses and I, too, agree they should receive the necessary attention.

That the turbinals have been abused there is no doubt. They are getting a rest since the submucous operation has been devised. But not all submucous operations are successful either. The constant dripping from the nose with repeated colds that oftentimes are blamed on the turbinals and deviated septum will often be cured by autogenous vaccines and save the patient from an operation.

Submucous operations have now gone beyond their line of safety and will gradually recede to their proper sphere, and the diseased polypoid terminals will again be removed often giving greater relief than the now overworked septal resection.

DR. JOSEPH BECK, Chicago: I think such a large subject as has been presented this morning should be discussed, if possible, from experience in some of the important questions that have been brought up. Referring to the first paper on chorea; in operating on something like fifty or one hundred cases, most of them referred by competent neurologists, for the removal of tonsils, most of the patients have been markedly improved, but very few have been cured as rapidly as the cases reported by the essayists. The question of erythema nodosum should be mentioned in connection with chorea because frequently that lesion is presented in these cases of tonsillar infection with very slight choreic symptoms. I have been disappointed in the removal of tonsils in chorioretinitis and other eye lesions. I have one case of iritis in mind in which I removed the tonsils. Dr. H. Gradle, after a roentgen-ray examination, did an iridectomy and many of the teeth were removed but it still went on to a very destructive condition of the eye. The question of indican interested me because of the work that is being done at the Mayo Foundation along that line. The substance seems to be an indican-iodine combination which is markedly diminished throughout the whole system by the removal of the particular focus of the infection, especially tonsils. In regard to the teeth, my experience is that the roentgen-ray is often interpreted by incompetent men; the teeth are removed and the question is not solved. In reference to skin lesions, I have had a number of cases, particularly psoriasis in which I removed the tonsils, but not until the vaccine was used that we prepared from the tonsillar excretion did results appear. As to after-treatment, I am of the opinion

that in the operation which we all try to do we are removing a physiologic structure, namely, the capsule; until we have perfected an operation that leaves the capsule we are going to have contractions and adhesions. As to iodine used after the operation, I have been using it for a time although I must admit there is some pain when it is applied. I seldom use it immediately after the operation, usually the following two or three days. What interested me particularly in Dr. Wright's paper is the question of these neuralgic pains and to relieve them an operation is frequently performed. But after everything is done they still come to us with their pains.

DR. GEORGE F. KEIPER, La Fayette, Ind.: I wish to speak a little from experience in several cases, especially one well-marked one in our hospital where after a careful removal of the tonsils we had a beautiful case of purulent infection of the knee joint. This led me to take this stand, that we make a culture from every case of tonsils removed so that if we need an autogenous vaccine we have the culture ready for use. Now as the iodine in the tonsil fossa, my patients complain very bitterly of pain, from the application of tincture of iodine. If you will use Lugol's solution you have no trouble with pain. Beware of the mouth that shines brilliantly with gold crowns. A careful roentgen-ray plate in those cases is indicated before we attempt a tonsil operation. The teeth may be harboring infection. With reference to the after-care of these cases, I have tried everything that has been suggested and have come to this conclusion, that the least we do the better, for this reason, if we are careful in checking the hemorrhage after tonsil removal we will not have very much trouble with our cases afterward because, as you know, a clot forms in the tonsillar fossa and the clot is composed of fibrin and blood corpuscles, and we need the sticky fibrin covering the wounded area to prevent the infection. All tonsil cases should be examined frequently for some time afterward, for in the removal of tonsils and adenoids we expose from 2 to 3 square inches of raw surface and that must heal over by granulation properly. Also, in these cases it is wise to pay some attention to the lingual tonsil for that also may be the cause of infection. Dr. Mayer has made a good point when he directs attention to the uvula.

DR. W. F. BEGGS, Newark, N. J.: Dr. Keiper in a measure has gone contrary to my idea in the method of after-treatment. I am only speaking from personal experience. If one makes a very careful dissection and has no oozing and no postoperative clot presents itself it needs very little, if any after-treatment. I have had two experiences in which postoperative clots became infected, and I feel that they produced secondary hemorrhages. A patient on whom I had performed a tonsillectomy returned with an aggravation of former symptoms. Examination revealed an infected organized blood clot which on removal cured up the case. Therefore, if you have this condition, it seems to me you favor a retention of the secretion, and a possibility of a recrudescence of the disease. Since using emetin hydrochlorid half an hour before operating and taking greater care to have my dissection perfect, I can truthfully say that the postoperative treatment is almost nil.

DR. WILLIAM S. TOMLIN, Indianapolis: The subject of tonsils at our meetings is always a matter of interest, for usually some good points are added to our previous papers. Focal infections are rarely single. Occasionally they start singly but do not continue so, and when we operate on a tonsil or puncture some ethmoid cells without a clear conception of the whole area to be examined we are failing to measure up to the standard of the specialist. I do not believe in diagnosis by exclu-

sion. If the tonsil is diseased it should be removed, a suppurating antrum should be opened up and diseased ethmoids should be removed. It has been the custom in the last few years to operate on a great many more cases in the acute stage of these focal infections. I have gone to the patient's bedside and removed them under local anesthesia when the patients were too ill to come to my office or to a hospital. Let us not be too sanguine or promise too much in the beginning. These cases of tonsillar infection in my judgment are the most serious when the infection is extracapsular. The tendency to absorption of the toxins as well as the production of bacteria proceeds more frequently from the peritonsillar inflammation. In these cases, in operations, pressure should not be made without previously making room for drainage. Make an incision at the plica so that when pressure is made on the tonsil there is room for egress for the infected discharge.

DR. H. V. DUTROW, Dayton, Ohio: I think it is a well established fact that great results have been obtained by the removal of tonsils and radical, up-to-date care of the teeth. I also think the two go hand in hand and, as one of the men present just remarked, "Beware of the shining gold crown." In practically every case of this kind I insist on a roentgen-ray examination. Most of you have heard Fisher of Cincinnati give his interesting talks on how the dental surgeon should proceed with his work. He condemns absolutely the full crown and advocates a jacket or half crown. I think a great many of us in our tonsil work very often slip a cog in our examination of the tonsils. Unless we do this very thoroughly we are going to overlook many diseased conditions of the tonsils that are causing the trouble for which the patient comes to us. I have been rather thorough in this examination recently, and my thoroughness has been impressed on me by the pathologic condition I found in the tonsils at the time of enucleation. Just recently a young girl of 13 or 14 years came to my office with a polyarthritis of the right arm. I examined her throat and tonsils, and said to her parents, "I cannot see very much the matter with her tonsils." I could not get any pus from the crypts (I have never used a transilluminator). I advised an immediate tonsillectomy, to which they consented. On enucleating the right tonsil fully half a drachm of pus exuded. The case made an uneventful recovery. There was no outward indication of this pocket of pus. Whether the transilluminator would have disclosed its presence I am not in a position to say. I should think, however, if transillumination has any practical value it would be in such a case.

We who do nose and throat work in conjunction with our eye work see a great advantage in attention to focal infections in chorioretinitis, and I am happy to say that we are now able to get good results in cases which formerly went on to blindness and even loss of the globe.

You all know of the recent interesting work of de Schweinitz with reference to the etiology of iritis and uveitis. Formerly syphilis had first place in the list of causative factors in these conditions, with rheumatism second. Now I think the position is reversed.

In view of Dr. Dabney's statement of loss of life due to infected tonsils, why should he deprecate the idea of their removal? Can he give any concrete instances of bad results following what might have been termed an unnecessary tonsillectomy? I dare say there are men in this room who have had their tonsils removed, and I should like to know if there is a single one who would like to have them replaced. Do you know among your practice of a single case that you feel that any damage has been done the patient by a properly executed tonsillec-

tomy? One gentleman answers, Yes. We should know of these cases and of the bad results in detail. It is in such a meeting as this where the exchange of views is most beneficial, and it should be free and frank.

DR. THOMAS E. CARMODY, Denver: I will try to cover these points as quickly as possible. I will begin with Dr. Wright's paper first. If you will refer your cases to the orthodontist, you will not have to operate. He speaks of pneumonia and pulmonary abscess following tonsil operations. I believe they will be less frequently found if such an apparatus is used. Some one has spoken of destruction of the plica and pillar. I do not believe in incision into the plica. I believe in incision at the edge of the plica. Dr. Keiper speaks of a clot in the fossa. I think he means clots in the blood vessels. In focal infections we should keep in mind Betts' formula, 50 per cent. infection and 50 per cent. immunity; so if we remove the tonsil very frequently we will overcome the rest of the infection by the immunity of the body. I have had personal experience with pain following tonsil operations and I use in many of these cases equal parts of orthoform and aristol. Another thing in the after-treatment is the use of hot boracic acid solution before the patient leaves the table. Syringe out the nose and throat. As to the use of iodine, I think the doctor made a mistake. We do not use 25 per cent. iodine. Lugol's solution helps a great deal. Now as to the teeth. My experience is that even where good dentistry has been done abscesses are frequently found at the apex. In one case in which I had a roentgenogram made I found only three teeth that did not have an abscess on them.

DR. CHARLES H. BAKER, Bay City, Mich.: I have great sympathy for the student who was going to remove all tonsils that he could see. I think his position was that the removal of a normal tonsil was no detriment to the person losing it. My friend, Dr. Perry of Seattle, calls the tonsils "a gland with a hare-lip." The tonsils belong to a system of lymph glands which are so numerous in the body that the loss of the one that is defective could be no loss to the system at large. The first speaker spoke of a very severe case of chorea cured by the removal of tonsils. I would call attention to the fact that not all those very severe choreas are of tonsillar origin but may be of refractive origin. I had a patient showing extreme general muscular spasm. I made a rough diagnosis of hyperopia. The boy was so nervous he could not hold still for a satisfactory examination. I dilated the pupils and with the shadow test made a tentative diagnosis of 2 diopters of hyperopia. In a week's time the boy was able to sit still for a very satisfactory examination. At the end of a year he came back having lost his glasses to have them replaced. He was a perfectly normal boy and had advanced two grades within the year. Now, that is mentioned not to say that I am opposed to the idea of the tonsillar origin of some of these cases but to show that there are other causes of these severe choreas.

DR. B. F. HODSON, Miami, Fla.: In the July number of the *Laryngoscope* there appeared an article on post tonsillectomy abscesses of the lung. There were nine cases reported at the Mt. Sinai Hospital, New York, and the diagnosis made by Dr. Morris Manges. These patients were not operated on at that hospital but were taken there because of symptoms simulating pneumonia which appeared two to three weeks following the operation. Such a calamity following the operation of tonsillectomy should cause some concern and warrant an investigation, and to our mind we have not far to look. All of these patients were adults and all were given a general anesthetic, probably ether. Was the pneumonia the result of bronchial irritation caused by ether? No, not per se, but by microorganisms squeezed out of the tonsil into the tide of mucus and

carried down the bronchial tree to a favorable spot for propagation. There is absolutely no more need of administering a general anesthetic to an adult for tonsillectomy than there is for a senile cataract.

DR. J. CLARENCE KEELER, Philadelphia: One prominent and distressing symptom which has not been mentioned, either in this excellent symposium or in the discussion on the tonsils, is that of toxic labyrinthitis. A patient frequently complains of dizziness, so great is the vertigo, at times, as to compel him to sit or lie down to prevent his falling. By a neuro-otologic examination, we have found the trouble to be in the labyrinth. To illustrate the point, I shall take the liberty to mention a case in our clinic at the Jefferson Hospital. Mrs. B. suffered such violent attacks of vertigo that she was compelled to lie in bed. These attacks occurred over a period of several years. Hoping to be relieved, she submitted to the removal of her ovaries and appendix. Still she was not relieved. Finally, she was referred to the Hospital with the diagnosis of a brain abscess. A thorough examination was made when both tonsils were found to be extremely hypertrophied and diseased. They were removed, and she made a perfect recovery which has extended over a period of two years.

DR. J. M. INGERSOLL, Cleveland: In regard to chorea and its relation to pathologic tonsils I have seen a fair number of cases of this sort. In about 80 per cent. removal of the tonsils has been beneficial. Naturally the laryngologist sees cases of chorea with pathologic tonsils, but of course there are other sources of infection. I talked with Dr. Dabney after the close of the meeting this morning. I rather got the impression that he made a practice of irrigating the nasal fossa after adenoid operations, but he corrected that impression. He does it only in selected cases with a very definite end in view. Personally, I do not believe in doing such a thing, on account of the danger to the ears and also because I think that it is better to do nothing to the nasopharynx after an adenotomy unless it is absolutely necessary. I want to emphasize what Dr. King has said in regard to thoroughness in diagnosis, and the point that Dr. Mayer made in regard to standardizing our methods of examination. Dr. Wright's paper was so good in his advocacy of conservatism, one needs scarcely say anything more except to agree with him and emphasize the points he makes. The conclusion to be drawn from all of the papers is I think careful, thorough and scientific observation in conjunction with good team work with the hospital interns.

DR. E. H. THOMPSON, Cincinnati: This morning one of the essayists took exception to operating too early on small children. I agree with him, but he did not go into detail for his reasons. This was forcibly brought home to me about five years ago by operating on a nursing child. It was necessary to give the child relief by removal of tonsils and adenoids. After the operation the mother came back to me and complained that the child in nursing would gag. It was hard to account for this. So it led me to observe a little more closely and as my own babies came into the world I watched very carefully. I came to the conclusion that it was a mistake to remove the tonsils of a nursing child for the reason that the tonsil has a distinct function in the nursing child. In the first place the tonsils stimulate the breast of the mother. As we know the breast is made of erectile tissue and the child in nursing takes the breast far back into the throat so that the nipple really rests on the two tonsils and in the act of swallowing these two tonsils stimulate the breast thereby producing an erection of the breast and this stimulation produces the secretion which is really necessary in the proper

function of the breast. The tonsils being less sensitive than the rest of the throat I believe it acts as a protection in a way. In early infancy I think it advisable to simply remove the adenoids and later to remove the tonsils.

DR. A. A. HAYDEN, Chicago: I have listened with intense interest to all this discussion and agree with some of it, and with some I heartily disagree. In regard to indications for removal of tonsils, I think there are only two. They should be removed when they obstruct and when they cause trouble by absorption. The former one is very easy to determine, the latter is hard, and the services of a competent internist and his well equipped laboratory must be called on to determine this question. I have no children myself and have never heard of the tonsil stimulating the erectile tissue of the mammary gland, but in my experience the indications are not subject to age limits at all. In regard to lung abscesses, I think that if more of us would use the excellent suction ether machine of Beck, lung abscesses would scarcely occur at all. In an experience of about 8,000 cases I have never seen a lung abscess. In regard to chorea and eye lesions, I believe that tonsillec-tomy on the advice of your internist should be a routine procedure. In regard to eye lesions, I was sorry to hear Dr. Beck say that he was not very enthusiastic about the relief which the removal of tonsils afforded. I have had some very startling improvement in cases of iritis. In regard to blowing out the caseous material as suggested by Dr. Mayer, I think there is some danger in this. In regard to after-treatment, my experience has been that if the dissection is well done, the after-treatment makes very little difference. In regard to operating on the nose and throat, there is more reaction but it is sometimes convenient to do the two especially in the dispensary cases. I believe very serious ear complications attend syringing the nose and throat. As to leaving a clot in the tonsil fossa, I believe that any visible hematoma is very apt to be the site of a hemorrhage.

DR. O. DULANEY, Dyersburg, Tenn. (closing discussion): I would not have this intelligent body of men understand that I mean all cases of chorea are due to infected tonsils. I merely reported a series of fourteen cases in which the pathology was found to be infected tonsils. I have seen the nodular form of erythema that was not due to tonsillar trouble at all. I have seen cases of chorea in which we could not find the pathology of it at all. But I have merely reported these cases, all of which presented well-defined choreic symptoms. Now, Dr. Motley, who is associated with me is an able internist. He does not do any general practice at all nor any work outside the hospital. These cases were referred to us by general practitioners and every possible part of the body was examined for focal infections. I say to my patients: "We are just beginning. We are just getting ready to treat our cases." And then by the association of your pathologist and the general practitioner you are able to cooperate with each other and get the best results in the treatment of these cases. I say to them that they must return for two years and be under my observation. I had one patient I know of in which case I had removed the tonsils and some slight symptoms were again returning three or four months afterward and I advised that a middle turbinectomy be performed. The turbinated bodies seemed to have the same infection as the tonsil, and also the lymphoid tissue follicles in the fauces contained the same infection as the tonsil, and must be treated to effect a cure. You cannot cure these cases without systemic treatment afterward, so I cannot say, and no other man can say, all cases of chorea are due to infected tonsils, but I think it bears some

relation to infections such as endocarditis, pericarditis, myocarditis or rheumatism. I think in the majority of cases it is an infection whether of the tonsil or some other part of the body.

DR. JAMES J. KING, New York: I thank you for your kindly and generous discussion of my paper. Dr. Mayer made a splendid point about standardizing the examination of the tonsils. In my practice I go through a regular routine. I take a culture, inspect the glands of the neck and inspect the tonsils together with the history of the case. Dr. Mayer's point about the uvula is well taken. I also agree with what he said about diagnosticians of ability. These cases too frequently pass through the hands of men of the greatest repute who never look into the mouth. I have had patients to go through the hands of half a dozen men and their tonsils have never been examined. I believe that every symptom has some basis of fact in pathology. If you find and remove that pathologic lesion you will cure the patient. Dr. Keiper stated that the lymphoid tissue at the base of the tongue has not been mentioned. I wish to remind him that I called attention to lymphoid tissue at the base of the tongue, and adenoid as well as tonsillar tissue. I am a great enthusiast about the use of vaccines, but in coming to you today I would rather underestimate the results than overestimate them. When I said that vaccines have produced remarkable results in suitable cases, it is a mild statement compared with what I actually feel after the experience I have had in many cases. I think the cases that are especially suitable for vaccine therapy are those which show a very low polymorphonuclear count. In many cases where this has been down to thirty and fifty they respond very rapidly. I had a patient whose polymorphonuclear count was down to about twenty-four and the small lymphocytes up to fifty-seven. Five injections of autogenous vaccines just reversed that blood picture. We have frequently seen patients get well with vaccines when everything else has failed. I have seen several cases of scleritis and episcleritis treated without results by the eye men, get well promptly by clearing up the tonsillar infection.

DR. JOSEPH W. WRIGHT, Indianapolis: One thing I would like to add; very often cases come to us from the man in general practice, and for some reason or other we hesitate to doubt his diagnosis. I have no hesitation in telling the man who refers the case. I believe very firmly that the man who takes tonsils out promiscuously is not doing proper scientific work. I recently heard of an institution in our state where 100 orphan children had their tonsils removed, and one general surgeon suggested that he go through with the appendices. I want to say to Drs. Carmody and Hayden that we use the suction apparatus of Dr. Beck, and that the case of abscess which I mentioned was supposed to have been an embolus and it was done under a general anesthetic.

THE PROGNOSIS AND TREATMENT OF TUBERCULOUS LESIONS OF THE LARYNX*

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Tuberculosis of the larynx is invariably secondary to tuberculosis of the lungs and should therefore be considered as a complication of the latter. The prognosis in laryngo-pulmonary tuberculosis depends not only on the condition of the larynx and lungs, but also on all other elements that may have a modifying influence on the patient's condition. Only after a careful consideration of all factors concerned may we hope to arrive at an accurate prognosis. The prognosis may be discussed in regard to (1) life and (2) voice. As a rule both are intimately connected, except in chronic cases, in which the voice may remain affected while the length of the patient's life is generally not influenced.

DETERMINING THE PROGNOSIS

The factors that determine the prognosis as to life are:

(1) *The Pulmonary Condition.*—The character and extent of the pulmonary lesion should be carefully studied. The chronic type of pulmonary tuberculosis is the most favorable and the outlook here is good, while in the acute and subacute cases the prognosis is more serious. Patients with the most extensive lesions do not give the poorest prognosis. One with a widely spread chronic fibroid lesion invariably offers a better prognosis than a patient with a limited subacute or acute lesion. In other words it is not so much the extent, as the character of the lesion that determines the gravity of the individual case. Of course a smaller lesion is of more favorable outlook than a more extensive involvement of the same type.

(2) *General Condition.*—Temperature, pulse and weight constitute the keynote to the patient's general condition. A rapid pulse usually signifies myocardial degeneration caused by the toxemia from the tubercle, and when present for a prolonged period renders the prognosis grave. Daily or frequent elevation of temperature for a considerable length of time shows that the case is either of the acute or subacute type, depending on the

*From the Municipal Sanatorium, Bureau of Hospitals, Department of Health, City of New York.

height of the fever. The chronic cases often have febrile exacerbations, which, however, last only a brief time. A continued sub-normal temperature is a bad prognostic sign. The patient's state of nutrition is also an important guide in the prognosis.

(3) *Underlying Disease, such as Gastro-Enteritis, Has a Marked Effect on the Prognosis.*—Proper digestion and assimilation are of valuable aid to the tuberculous individual, as nothing will have a more deleterious effect on the patient's general condition than a disordered digestion. A good appetite therefore is indispensable to recovery and for this reason is an important guide in the prognosis of laryngo-pulmonary tuberculosis. Syphilis undoubtedly aggravates the prognosis by lowering the general vitality of the patient.

(4) *Type of Laryngeal Lesion.*—As previously pointed out by the author¹ there are three distinct types of tuberculous laryngitis, namely, the acute, subacute, and chronic. There is also a peracute type, in which the condition is hopeless and the length of the patient's life is usually from a few weeks to three months. The prognosis in the acute cases is more favorable than in the peracute, and we occasionally see such cases heal. In the subacute cases the prognosis is much better. This is the type that is most amenable to treatment, and when other factors are favorable the prognosis is good. The chronic cases are the most hopeful. Here the voice may remain affected, but the length of the patient's life is generally not influenced. We must, however, always bear in mind that any one of the lesions may suddenly change its course, and for this reason the prognosis should always be guarded.

(5) *Location of the Lesion.*—The prognosis as to life is most favorable in cases in which the lesion is situated in the posterior commissure or on the true cords. Such lesions usually are of the subacute or chronic type and tend to heal by fibrosis. Lesions of the epiglottis, aryepiglottidean folds, arytenoids, and ventricular bands may be of the acute, subacute or chronic type, and the outlook here is less favorable. In other words, when the mucous membrane is closely adherent, such as on the true cords and posterior commissure, the disease takes a chronic course and the prognosis is therefore favorable. When the mucous membrane is loosely adherent, such as on the epiglottis, folds, arytenoids and ventricular bands, the course is usually more acute and the prognosis less favorable.

1. Dworetzky, J.: Clinical Pathology of Tuberculous Laryngitis. Ann. Otol., Rhin. and Laryng. December, 1916, p. 804.

(6) *The Extent of the Lesion.*—This does not necessarily influence the prognosis as to life, although cases of slight involvement are the most hopeful ones as far as the voice is concerned; nor is the prognosis less favorable in cases with ulceration. In fact, local medication takes better effect when ulcers are present. Thus we may say that widespread lesions, unless of the acute type, do not necessarily indicate a poor prognosis as to life.

(7) *Early Diagnosis.*—It is evident that the earlier the diagnosis is made the more easily can the lesion be cured or checked in its progress. This applies especially to the subacute cases which are most benefited by treatment and also to the chronic cases. The local resistance in the chronic cases is naturally lowered. The patient is usually unaware of the condition, and by early diagnosis and proper instruction regarding the care of the larynx, a chronic lesion will often be prevented from becoming acute. The acute cases, which as a rule take their own course, are occasionally helped by an early diagnosis. The author recalls a number of acute cases that showed marked improvement after an early diagnosis was made and early treatment was instituted.

(8) *Early Treatment.*—What has been said of the early diagnosis may also be said of the early treatment. The sooner the proper treatment is instituted the more favorable is the prognosis.

(9) *Nasal Obstruction or Disease.*—Sinuitis, pharyngitis, or tonsillitis tend to lower the resistance of the larynx and thus influence the prognosis.

(10) *Financial Status.*—The economic condition of the patient is often an important factor. Tuberculosis is a protracted and tedious disease and it takes about three to five years to accomplish an arrest of the lesion. Ample financial resources are necessary to "cure" over such a prolonged period. It is evident that the patient of independent means, and one who is faithful to the treatment, offers a better prognosis than his fellow of insufficient financial resources, who, in order to obtain a livelihood, must return to business or manual labor after only a brief course of treatment.

In giving a prognosis as to life, therefore, all the above factors should be carefully studied. Although in a number of cases the lesion in the larynx runs a course independent of the lesion in the lung, in the great majority of the patients the laryngeal lesion serves as an indicator of the pulmonary condi-

tion; and an active process in the larynx generally indicates activity in the lungs. Not only is a thorough knowledge of all these factors necessary but the laryngeal lesion itself should be watched for a considerable length of time so that the course may be definitely determined. In cases in which the lesion in the larynx is the chief predominating factor and runs an independent course, the general prognosis will of course depend chiefly upon the condition in the larynx.

The prognosis as to voice is usually more definite. This will depend largely upon the extent, location and character of the local lesion. The more extensive the process the greater the probability that the voice will remain affected. Cases with involvement of the true cords and interarytenoid space give a poorer prognosis as to voice than if situated elsewhere in the larynx. The destruction of tissue is usually greatest in the acute and sub-acute cases, and there the voice suffers mostly. Of course the other factors, such as early diagnosis and treatment, influence the local as well as the general prognosis.

In general, then, it may be said that no definite prognosis as to life can be made from the condition in the larynx alone. All factors should be carefully considered and the laryngeal condition should only serve as an aid in the prognosis.

TREATMENT

The treatment of tuberculous laryngitis is (1) prophylactic; (2) general, and (3) local.

(1) *Prophylactic Treatment*.—This consists of the removal of predisposing factors capable of producing a catarrhal condition of the larynx with a consequent formation of a favorable site for the implantation of the tubercle bacillus. The predisposing factors are (a) nasal obstruction or disease; (b) pharyngitis and tonsillitis; (c) frequent colds; (d) abuse of the voice; (e) excessive cough, and (f) local irritants, such as smoke, dust, tobacco and alcohol. All these factors have a deleterious effect on the larynx and if possible should be removed.

At the Otisville Sanatorium each building has the following notice posted for the information and guidance of the patients.

“IMPORTANT.—Patients with pulmonary tuberculosis not infrequently develop tuberculosis of the larynx, but like tuberculosis elsewhere in the body, it can be prevented.

To prevent tuberculosis of the larynx, patients are advised:

(1) Always to breathe through the nose. Mouth breathing

is a bad habit. If you cannot breathe through your nose there is a cause which needs treatment.

(2) Refrain from abuse of voice, such as shouting, singing and excessive talking.

(3) Cough as little as possible. Coughing irritates the throat.

(4) Patients subject to "colds" or sore throat can often be helped by prompt medical attention.

(5) Abstain from the use of tobacco and alcohol. These act as direct irritants to the throat.

(6) Patients having the slightest trouble in the nose and throat should consult the doctor."

(2) *General Treatment.*—The general treatment is of primary importance. It is generally admitted that tuberculous laryngitis is invariably secondary to pulmonary tuberculosis, and by treating the larynx and disregarding the lungs we actually ignore the main issue. It is not only futile but also detrimental to a patient with symptoms of toxemia to go to the office or clinic for daily treatment of the laryngeal condition. The lungs should receive our first attention, and activity in the lungs requires absolute rest. The larynx in such cases may be treated with the patient in bed but never otherwise. Only cases with no activity in the chest may be allowed some exercise and may therefore be treated in the office, and in these cases, too, we must never overlook the pulmonary lesion. The sanatorium is the ideal place, although a number of patients that are financially able could be successfully treated at home. The country, where plenty of pure air can be obtained, is undoubtedly the most desirable place of residence. The dry mountainous climate is the most agreeable in the great majority of instances. Outdoor sleeping should be urged, and persistent "curing" on a porch with a southern exposure will often accomplish surprising results. The exercise should be well regulated, and prescribed as the activity in the lungs and larynx subsides. The diet should be full and nourishing, and we must always remember that good digestion is the greatest asset in tuberculous patients. The stomach should never be abused and the diet should be carefully watched and regulated. As a rule three good meals judiciously selected are sufficient. Nourishment between meals should be recommended to patients whose weight is not up to the standard, and who apparently benefit by it. The author feels that the general treatment of the patient is far more important than the local treatment, and a patient receiving

only local attention with half-hearted or no treatment for the pulmonary condition is not being properly treated.

(3) *Local Treatment.*—No specific remedy has as yet been discovered for tuberculosis of the larynx. The remedy, which we hope will eventually be discovered for the cure of tuberculosis of the lungs, will also cure the larynx. You are familiar with the different remedies tried such as formalin, lactic acid, argyrol, silver nitrate, iodoform, etc. The author has tried most of them and found some cases to be benefitted by one, some by another, while a good many improved without any treatment and healed spontaneously. The method of treatment employed at the Otisville Sanatorium and found to give the best results is as follows:

First determine whether the case is acute, subacute or chronic. If chronic and patient only suffers from slight discomfort, such as occasional tickling, irritation, etc., he is treated with a spray of 5 per cent. menthol in olive oil at frequent intervals. If no discomfort is present, he is told of his condition, warned not to abuse the larynx in any way and no local treatment is advised. The acute and subacute cases are treated locally at regular intervals. Such patients are supplied with De Vilbiss atomizers and told to spray the larynx four or five times a day with an alkaline solution, followed by a solution of 5 per cent. menthol in olive oil. If properly used the larynx is thus kept clear of accumulated muco-pus and the patient is relieved of much of the discomfort.

Rest of voice is imperative in the acute and subacute cases. Absolute silence for the acute cases, while the whispered voice may be allowed as the case gradually becomes subacute. The patients with lesions of the chronic type may use the voice without harm. Abuse of the voice should of course always be discouraged.

For topical application the remedy found to yield the best results is the solution of iodine in glycerin. It is of the greatest benefit in the subacute and also in some of the acute cases. The author uses it in strengths varying from 1.5 to 7.5 per cent. and applies it directly to the larynx on a laryngeal applicator. He begins with the 1.5 per cent. three times a week gradually increasing the strength as the patient develops a tolerance for the solution. The application should be made gently during quiet breathing, and the entire larynx may be covered with it by asking the patient to phonate while the applicator is in the larynx, thus squeezing the swab with the true and false

cords. The swab is then immediately removed and the patient is told to breathe quietly thus preventing a coughing spasm in the majority of instances. When the stronger solutions are used and especially in cases with fresh ulceration the patient often experiences sharp burning for a few seconds. This can easily be mitigated by previously spraying the larynx with a 2 per cent. solution of cocain. Under this treatment, the author has observed infiltration to diminish, granulations to disintegrate and absorb and ulcerations to heal. The solutions of iodine should be in brown bottles with glass stoppers. The action of the iodine on the lesion is probably due to its penetrating and antiseptic properties. The tubercle is avascular and cannot be reached by drugs through the circulation while iodine is known to have a special affinity for tuberculous tissue. The iodine may also possibly exert its beneficial effect by causing mild irritation thus producing slight reaction. The following are the formulas used:

Solution Iodin No. 1.

| | |
|----------------|--------------------------------|
| R Iodin Cryst. | gr. $\overline{\text{VII ss}}$ |
| K. I. | gr. $\overline{\text{XV}}$ |
| Glycerin qs. | oz. $\overline{\text{I}}$ |

Solution Iodin No. 2.

| | |
|----------------|-----------------------------|
| R Iodin Cryst. | gr. $\overline{\text{XV}}$ |
| K. I. | gr. $\overline{\text{XXX}}$ |
| Glycerin qs. | oz. $\overline{\text{I}}$ |

Solution Iodin No. 3.

| | |
|----------------|---------------------------------|
| R Iodin Cryst. | gr. $\overline{\text{XXII ss}}$ |
| K. I. | gr. $\overline{\text{XLV}}$ |
| Glycerin qs. | oz. $\overline{\text{I}}$ |

Solution Iodin No. 4.

| | |
|----------------|-----------------------------|
| R Iodin Cryst. | gr. $\overline{\text{XXX}}$ |
| K. I. | dr. $\overline{\text{I}}$ |
| Glycerin qs. | oz. $\overline{\text{I}}$ |

Solution Iodin No. 5.

| | |
|----------------|-----------------------------------|
| R Iodin Cryst. | gr. $\overline{\text{XXXVII ss}}$ |
| K. I. | gr. $\overline{\text{LXXV}}$ |
| Glycerin qs. | oz. $\overline{\text{I}}$ |

Cases with superimposed catarrhal laryngitis or tracheitis are distinctly benefited by the intratracheal injection of 5 per cent. menthol in olive oil at frequent intervals. It allays irritation, diminishes cough and facilitates expectoration.

For relief of pain in laryngeal tuberculosis orthoform or cocain before meals will usually enable the patient to eat his food without distress. When the dysphagia is apparently due to infiltration and ulceration of the epiglottis, even if the lesion is not entirely confined to the epiglottis, the patient will often be relieved of this troublesome symptom by an epiglottidectomy. This operation is absolutely indicated in cases where the lesion is limited to the epiglottis, and the patient's general condition permits.

SUMMARY

In summarizing the treatment of tuberculosis of the larynx we may say then that the most important measures are:

- (1) The local and general prophylaxis.
- (2) The general treatment.
- (3) Vocal rest in selected cases.
- (4) Topical applications with the solution of iodine in increasing strengths.

I wish to express my thanks to Miss Mary M. Nordt for valuable assistance, and to Mr. John Klement and Mr. Louis Schroeder, the artists, who made the drawings of the larynx.

REPORT OF CASES

CASE 1.—*Figure 1*.—E. Mc. H., aged 19, was admitted to the Otisville Sanatorium, July 20, 1916. Examination of lungs showed a moderately advanced lesion with a positive sputum. Larynx was found negative. Soon after admission patient began to show signs of activity as evidenced by slight febrile exacerbations in the afternoon, and a somewhat exaggerated pulse rate. These symptoms of activity, however, soon subsided. Nov. 18, 1916, patient complained of slight dysphagia and on examination it was found that the epiglottis was congested and infiltrated, and was diagnosed as acute tuberculosis of the larynx. The patient was then treated with local applications of iodine solution. Dec. 21, 1916 it was found that the right half of the epiglottis and the right aryepiglottidean fold were eroded. Jan. 9, 1917 the right arytenoid showed infiltration and ulceration, and later the entire right half of the larynx, including the posterior commissure, were involved. As the activity in the larynx increased the tuberculous process in the lungs became active also. The temperature rose daily to 101-102 F. Tachycardia became pronounced, and the patient's pulse rate was 110 to 140 while at absolute rest. During an ordinary chest

examination the pulse would rise to 180 per minute. During the first five months the patient showed a gain in weight of 16 pounds, while during the last four months she lost the entire amount gained. The patient left the sanatorium on April 8, 1917 unimproved, and died about three months later.

DISCUSSION

The outstanding features of this case are:

- (1) The patient lived for about eight months after the development of the laryngeal lesion.
- (2) The patient was doing well up to the time of development of the lesion in the larynx.
- (3) The pulmonary lesion became active soon after the laryngeal involvement was discovered.
- (4) The lesion began at the epiglottis and spread to the right fold and arytenoid and posterior commissure. Acute lesions beginning at the epiglottis usually are fatal.
- (5) In spite of the ulcerations there was never enough dysphagia to interfere with the taking of food.
- (6) The pronounced tachycardia, which is generally a grave prognostic sign.
- (7) Treatment did not have any modifying influence on the course of the disease.

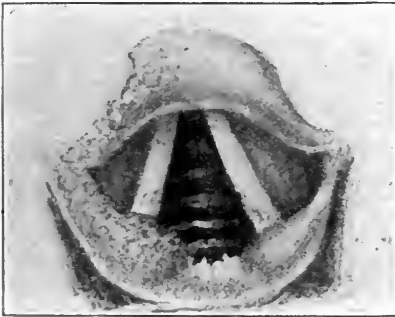


Fig. 1 (Case 1).—Infiltration of epiglottis with ulceration of the right half; infiltration and erosion of right ary-epiglottidean fold and arytenoid, and hyperplasia at posterior commissure. *Diagnosis*:—Acute tuberculosis of the larynx.

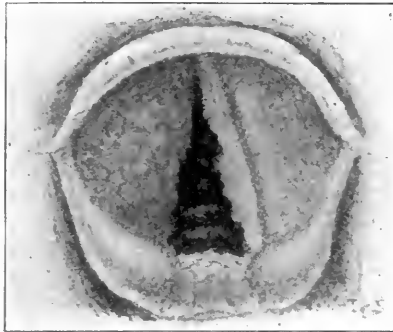


Fig. 2 (Case 2).—Erosions of entire right vocal cord with infiltration and erosion of right ventricular band. "Mouse-eaten" appearance of left cord with hyperplasia at posterior commissure and thickening of both arytenoids. *Diagnosis*:—Chronic tuberculosis of the larynx.

CASE 2.—*Figure 2.*—F. F., aged 28, was admitted to the Otisville Sanatorium, March 15, 1917 with advanced pulmonary tuberculosis. The entire right vocal cord was eroded, with infiltration and erosion of the right ventricular band. Also partial erosion ("mouse-eaten" appearance) of the left cord and hyperplasia at the posterior commissure with chronic infiltration of both arytenoids. Wassermann test negative. There was a history of hoarseness for past fifteen months with occasional aphonia. The onset of the hoarseness was gradual. The diagnosis was chronic tuberculosis of the larynx. Soon after admission patient developed complete aphonia and dysphonia

which persisted during his entire stay at the sanatorium. The larynx then showed acute edema of both arytenoids with complete erosion of both cords. The larynx did not respond to treatment and the patient left the sanatorium on June 28, 1917 and died Aug. 26, 1917.

DISCUSSION

This case illustrates that occasionally a chronic lesion will suddenly change its course and become acute. Such a course is uncommon but does happen, and for this reason the prognosis should always be guarded.

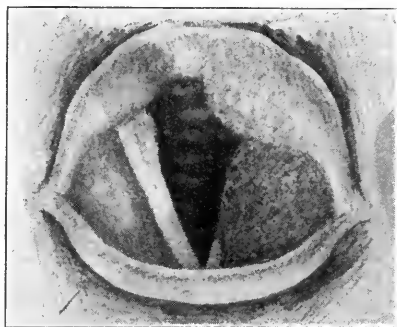


Fig. 3 (Case 3).—Edema and ulceration of right ventricular band and right arytenoid. *Diagnosis*.—Acute tuberculosis of the larynx.

CASE 3.—*Figure 3*.—P. V., aged 38, admitted to the Otisville Sanatorium, Feb. 23, 1916. On examination the pulmonary lesion was found moderately advanced with slight activity. Larynx showed edema and ulceration of the right ventricular band and the right arytenoid, also slight edema of the left arytenoid with infiltration of the vocal process. The Wassermann test was negative. Discomfort in the larynx was the principal subjective symptom. The diagnosis was acute tuberculosis of the larynx. In about one month after admission patient began to show improvement in the larynx. The examination on May 2 showed marked fibrosis of right ventricular band, and right arytenoid. Process becoming subacute. The local treatment that the patient received consisted of sprays and several applications of the solution of iodine. The patient was discharged from the sanatorium Jan. 14, 1917.

Sept. 25, 1917 in answer to an inquiry the patient wrote, saying that he has gained 10 pounds in weight since he left the sanatorium, and that his throat does not give him any trouble at all.

DISCUSSION

From the patient's statement it might be surmised that the process in the larynx has now become chronic and quiescent, thus illustrating that occasionally an acute case changes its course and will heal.

CASE 4.—*Figures 4 and 5.*—I. T., aged 20, admitted to the Otisville Sanatorium on December 28, 1916. Chest examination revealed a moderately advanced lesion of the lungs. On the third day at the sanatorium, the patient developed a slight afternoon fever which afterwards became persistent, rising to 101 and 102° F. on every afternoon, while the pulse ranged between 110 and 130 during his entire stay at the sanatorium. The examination of the larynx, on admission, showed a uniform infiltration of the epiglottis with slight thickening at the posterior

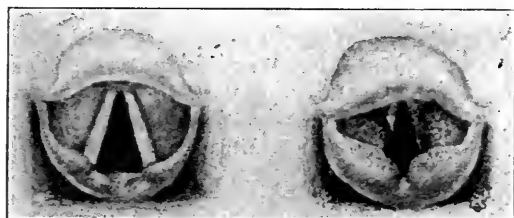


Fig. 4 (Case 4).—Acute edema of epiglottis; slight interarytenoid thickening.

Fig. 5 (Case 4).—Acute infiltration with ulceration of all parts of the larynx.

commissure and arytenoids (Fig. 4). January 5 the patient complained of slight dysphagia, and the examination showed the beginning of tuberculous infiltration of the oropharynx which kept getting more extensive, while the symptoms were gradually becoming more troublesome. February 1, the patient complained of severe dysphagia and regurgitation of liquid food through the nostrils. He then showed extensive infiltration and ulceration of the oropharynx and fauces. Uvula was edematous. Larynx showed edema of epiglottis and both arytenoids, and the Wassermann test was negative.

Feb. 14, 1917, the lesion was widespread. Uvula was much swollen and covered with macroscopic tubercles. The lingual tonsils were also ulcerated. The larynx showed acute edema with macroscopic tubercles of all parts (Fig. 5). All during the stay at the sanatorium the patient received local applications of solution of iodine to the affected parts, with no apparent benefit. February 15, the patient was transferred to a city hospital where he died a few days later.

DISCUSSION

The chief points of interest in this case are:

- (1) The patient died in about eight weeks after the diagnosis of the laryngeal lesion was made.
- (2) As the pulmonary lesion became acute the laryngeal involvement also became acute.
- (3) The lesion began at the epiglottis, and this should always be regarded as a bad sign.
- (4) The acute involvement of the posterior wall of the oropharynx, the fauces, the tonsils, the uvula and the lingual tonsils.

(5) The overwhelming toxemia as evidenced by the range of pulse and temperature.

This is a typical case of the peracute type and the prognosis is extremely grave.

CASE 5.—*Figure 6*.—P. S., aged 22, admitted to Otisville Sanatorium Oct. 28, 1915 and was in an advanced stage of phthisis. Examination of the larynx revealed infiltration with superficial ulceration of both ventricular bands, erosions of both cords and swelling of both arytenoids. About five months after admission the involvement in the larynx became acute, the epiglottis much infiltrated and eroded and the dysphagia pronounced. Amputation of the upper two-thirds of the epiglottis was followed by marked local and general improvement. The lesion became subacute and the patient was discharged from the sanatorium March 22, 1917, with the laryngeal lesion quiescent, and the pulmonary condition improved. Sept. 25, 1917, in answer to an inquiry the patient wrote saying that he made wonderful improvement in his general and throat conditions, and that the voice was surprisingly good, and that he feels much better than on the day of the discharge from the sanatorium.

DISCUSSION

The interesting feature of this case is the local and general improvement which followed the amputation of the epiglottis.



Fig. 6 (Case 5).—Acute lesion in the larynx with infiltration and erosion of epiglottis.

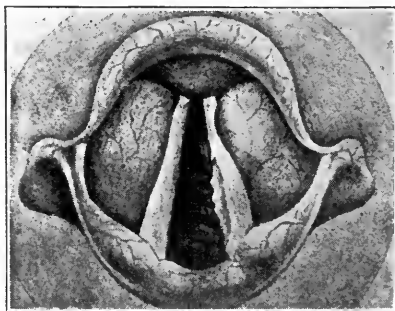


Fig. 7 (Case 6).—Infiltration and erosion of both cords, especially left with a longitudinal ulcer. *Diagnosis*.—Subacute tuberculosis of the larynx.

CASE 6.—*Figure 7*.—J. A., aged 24, admitted to Otisville Sanatorium, June 25, 1914. Examination revealed a moderately advanced lesion with bacilli in the sputum. Larynx revealed infiltration and erosion of both cords, especially the left which showed a longitudinal ulcer. Infiltration of both ventricular bands partially covering the true cords. Patient suffered from marked hoarseness, and lesion having passed an acute stage was undergoing fibrosis. The diagnosis then was subacute tuberculosis of the larynx. Sept. 9, 1915, the examination showed

marked infiltration with ulceration of both cords while other findings were same. The patient was discharged from the sanatorium, Oct. 7, 1915, both larynx and lungs unimproved.

In answer to an inquiry his family recently wrote saying that the patient died Nov. 19, 1916.

DISCUSSION

Patient lived for about two and a half years after initial diagnosis of laryngeal tuberculosis. The lesion apparently began as acute, a little later showed some tendency to fibrosis, and finally became acute again.

CASE 7.—*Figures 8 and 9.*—P. F., aged 21. was admitted to Otisville Sanatorium Sept. 21, 1916 and complained of hoarseness. A broad base papillomatous growth was found in the posterior commissure of the larynx. It represented the characteristic rugged appearance with adhering streaks of mucopus,

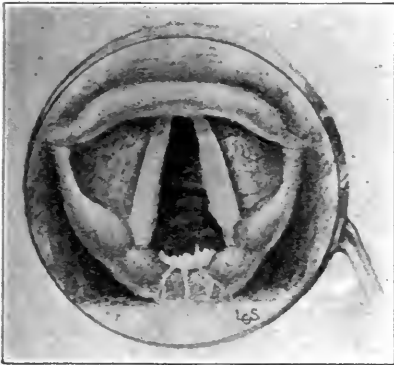


Fig. 8 (Case 7).—Broad base papillomatous growth at posterior commissure. *Diagnosis:*—Subacute tuberculosis of the larynx.

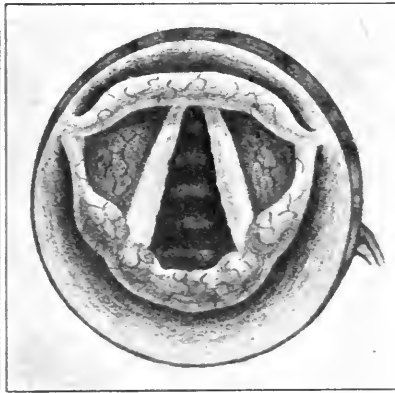


Fig. 9 (Case 7).—Same patient after seven months of treatment. Papilloma has disappeared.

and there was infiltration and slight ulceration of the free edges of both true vocal cords (Fig. 8). The patient was in an advanced stage of pulmonary tuberculosis. The diagnosis was subacute tuberculosis of the larynx.

Figure 9 shows same patient as in Figure 8, after seven months of local treatment with the iodine solution in increasing strengths. The examination showed that the papillomatous infiltration at the posterior commissure had entirely disappeared leaving some slight scar tissue. The cords are still somewhat thickened and slightly rough at the free edges. Hoarseness very slight. No discomfort.

DISCUSSION

The interesting features in this case are:

- (1) The beneficial effects of the iodine solution on the papillomatous growth.
- (2) The lesion which was previously subacute is now chronic.

CASE 8.—*Figures 10 and 11.*—J. C., aged 32, first admitted to the Otisville Sanatorium, Aug. 21, 1913. The examination of the chest revealed an early pulmonary lesion with a positive sputum. The larynx showed papillomatous infiltration at posterior commissure. Slight thickening of left arytenoid and cartilage of Wrisberg. Both cords slightly infiltrated and fibrosed (Fig. 10). Constant dryness and irritation in larynx. Marked hoarseness for past three years, the latter mostly due to the incomplete approximation of the cords. The diagnosis was subacute tuberculosis of the larynx. About two years after admission the patient was found to give a ++++ Wassermann reaction.

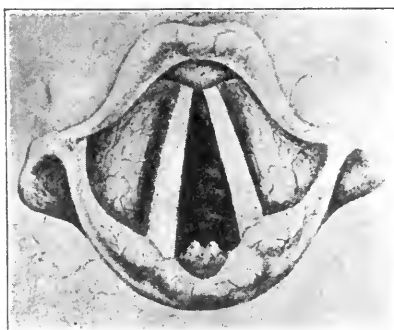


Fig. 10 (Case 8).—Papillomatous infiltration at posterior commissure. *Diagnosis:*—Subacute tuberculosis of the larynx.

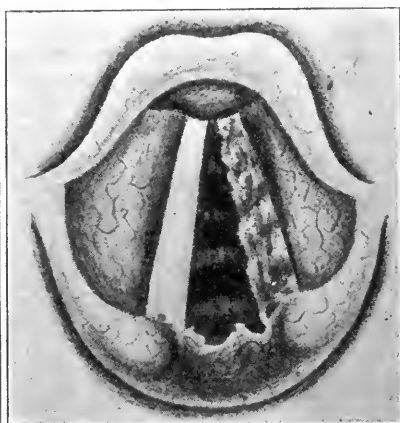


Fig. 11 (Case 8).—Same patient as in figure ten about three years later. Infiltration of left cord; papillomatous infiltration and ulceration at the posterior commissure.

mann reaction. There was no history of an initial lesion. He was then put on mixed treatment and given four intravenous injections of salvarsan, at the end of which time the reaction was still positive and the condition in the larynx remained about same. April 1, 1916, the patient left the sanatorium and received intensive antisyphilitic treatment in a New York City clinic. This treatment extended over a period of six months and consisted of fourteen intravenous injections of salvarsan in addition to the mixed treatment. The patient was readmitted to the sanatorium, June 21, 1917. The examination then revealed a far advanced pulmonary lesion with moderate amount of activity. Sputum positive. The larynx showed infiltration of left cord and papillomatous infiltration and ulceration at posterior commissure (Fig. 11). Marked hoarseness, slight dysphagia. Wassermann reaction was negative. Three months later another Wassermann test was made which, however, proved positive.

DISCUSSION ON DIAGNOSIS AND PROGNOSIS

The laryngeal lesion has all the ear marks of tuberculosis and the original diagnosis is likely correct. The positive Wassermann may be

indicative of a syphilitic infection in spite of a negative history. The prognosis in this patient is uncertain on account of the different elements entering into the case.

CASE 9.—*Figure 12.*—J. C., aged 38, admitted to the Otisville Sanatorium in 1913 and was found to have a moderately advanced pulmonary lesion with a positive sputum. Wassermann negative. Larynx showed a tuberculoma of interarytenoid space with healed erosions. Thickening and infiltration of both cords, especially the left. Left cord also showed healed ulceration on its upper surface. Infiltration of both ventricular bands, especially the right, which almost entirely covered the right cord (Fig. 12). Lesion showed complete fibrosis, and outside of marked hoarseness, patient had no discomfort. The diagnosis was chronic tuberculosis of the larynx.

Examination of larynx three and one-half years later showed the findings to be identically the same. In the meantime the patient has been doing very well, and shows no signs or symptoms of decline. The probable duration of the lesion according to the patient's history is about ten years. The patient does not receive any local treatment.

DISCUSSION

This case illustrates:

- (1) That an extensive lesion does not always indicate a poor prognosis. This lesion being widespread.
- (2) That the laryngeal lesion has already existed for about ten years, without harming the patient.
- (3) That the prognosis as to life will likely remain uninfluenced by the laryngeal lesion, while the voice will remain affected.
- (4) That no local treatment is necessary in such cases.

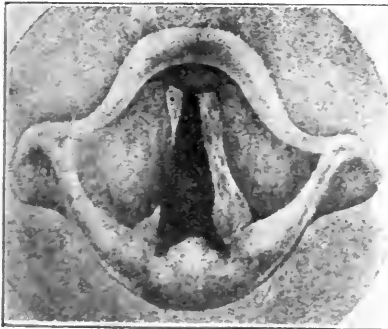


Fig. 12 (Case 9).—Large tuberculoma at posterior commissure with infiltration and fibrosis of nearly all parts of the larynx. *Diagnosis:*—Chronic tuberculosis of the larynx.

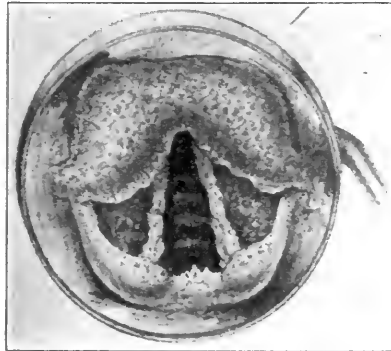


Fig. 13 (Case 10).—Warty growth of all parts of the larynx. *Diagnosis:*—Lupus of the larynx.

CASE 10.—*Figures 13 and 14.*—E. C., aged 24, admitted to the Otisville Sanatorium Jan. 11, 1917. Sputum negative. Wassermann reaction negative. Physical examination of chest revealed no signs of pulmonary involvement. Lungs also nega-

tive on roentgen-ray examination. Larynx showed a warty growth of all parts (Fig. 13). Hoarseness, dysphonia and discomfort in the larynx were the troublesome symptoms. The diagnosis was lupus of the larynx.

Figure 14 shows the same patient as in Figure 13, about seven months later. The cords and arytenoids were remarkably clear. The lupoid growth was limited to the epiglottis. The only treatment the patient received in addition to the general treatment consisted of local applications of argyrol, iodine solution and intratracheal injections of menthol in olive oil.

DISCUSSION

It is remarkable to note how such an extensive lupoid lesion cleared up in a comparatively short period and without any surgical treatment.

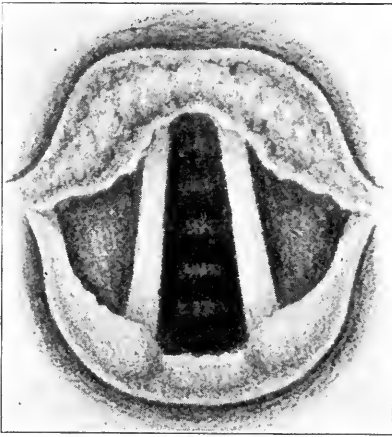


Fig. 14 (Case 10).—Same patient as in Fig. 13 seven months later.

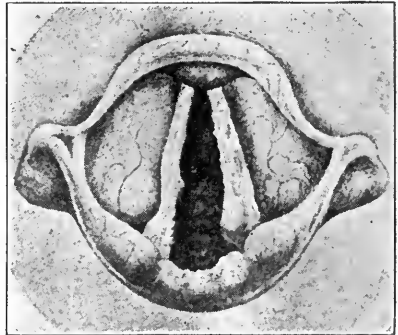


Fig. 15 (Case 11).—Marked infiltration and ulceration of both cords. *Diagnosis:*—Subacute tuberculosis of the larynx with tendency to fibrosis.

CASE 11.—*Figures 15 and 16.*—J. C., aged 23, admitted to the Otisville Sanatorium, April 22, 1915, and on examination pulmonary lesion was found moderately advanced. Sputum positive. Larynx showed marked infiltration and ulceration of both cords, presenting a nibbled out appearance. Interarytenoid hyperplasia with wrinkling of mucous membrane. Slight edema of right ventricular band (Fig. 15). History of dysphagia and marked hoarseness for past one and one-half years. The diagnosis was subacute tuberculosis of the larynx. Several months later the patient showed distinct fibrotic changes in larynx, and was discharged from the sanatorium, Aug. 3, 1916 with larynx markedly improved.

Figure 16 shows same patient as in Figure 15, who was readmitted to Otisville Sanatorium, Aug. 13, 1917, and the examination revealed infiltration of right arytenoid and fold, right half of epiglottis, and posterior portion of right cord. The infiltration was of the chronic granular type, and looked

almost like a lupoid growth. The left cord and anterior portion of right cord all cleared up (Fig. 16). There was still a moderate amount of hoarseness. Wassermann reaction negative.

DISCUSSION

The change of the subacute course into the chronic renders the prognosis in this patient favorable.

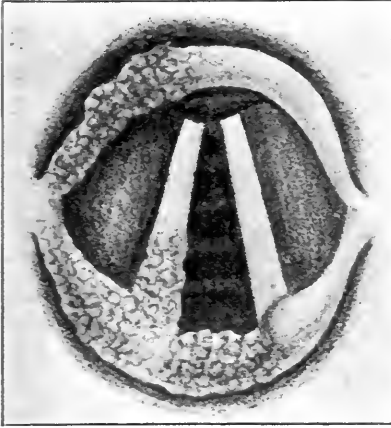


Fig. 16 (Case 11).—Same patient as in Fig. 15 one year later. Infiltration of right half of larynx, resembling a lupoid growth.

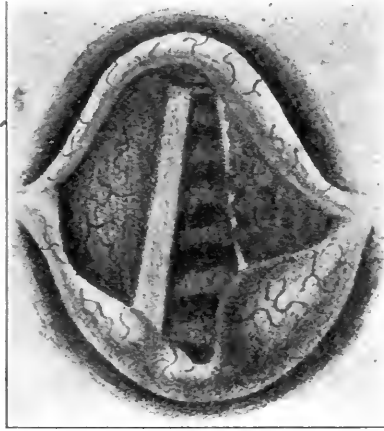


Fig. 17 (Case 12).—Pear shaped infiltration of left arytenoid and ary-epiglottidean fold. Infiltration with erosions of left ventricular band.

CASE 12.—*Figure 17.*—M. B., aged 20, admitted to the Otisville Sanatorium, May 17, 1917. Sputum positive. Wassermann reaction negative. The examination of the chest revealed a moderately advanced lesion with slight activity. The laryngeal examination showed pear-shaped infiltration of left arytenoid and aryepiglottidean fold. Infiltration and erosion of left ventricular band. Infiltration of left half of epiglottis which is drawn to the arytenoid (Fig. 17). Patient suffered from slight hoarseness and occasional discomfort in larynx. No other symptoms. Lesion running a subacute course. In addition to the general treatment this patient has received local applications of iodine solution for past five months with marked improvement. Swelling much diminished and symptoms disappeared.

DISCUSSION

This patient has benefited much from local treatment with iodine solution the lesion showing a tendency to fibrosis.

CASE 13.—*Figure 18.*—D. A., aged 30, was admitted to the Otisville Sanatorium, July 16, 1917. Sputum positive. Wassermann reaction negative. Pulmonary lesion moderately advanced with slight activity. Larynx showed infiltration of posterior commissure and thickening of both arytenoids. Infiltration with erosion of left ventricular band, covering the entire left cord

(Fig. 18). Patient has had marked hoarseness for past nine months.

DISCUSSION ON PROGNOSIS

With the laryngeal lesion of the chronic type and well fibrosed, and with the pulmonary lesion healing the prognosis is very good as to life, while the voice will likely remain unimproved.

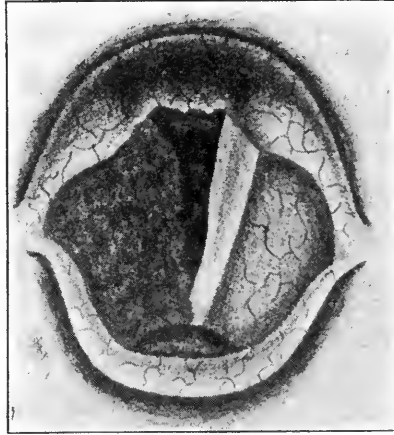


Fig. 18 (Case 13).—Infiltration with ulceration of left band, and interarytenoid hyperplasia. Lesion is of the chronic type and well fibrosed.

DISCUSSION

DR. EMIL MAYER, New York: Dr. Dworetzky's paper has been thorough as usual, and shows the result of a most careful investigation, but he speaks from the standpoint of the man who has seen many cases in a sanatorium. I want to talk to you from our own standpoint, and for that reason I want to take exception to the very first line of his statement, that laryngeal tuberculosis is the result of pulmonary tuberculosis. We have lots of cases of primary tuberculosis of the larynx, and we must also accept the lupus cases as tubercular. I am on record as having shown a case of lupus of the larynx that I treated 18 years before pulmonary complication set in. Nor would I say, in my own opinion, is the presence of tuberculosis of the epiglottis less favorable than in other parts. The fact that Dr. Dworetzky himself has shown you a splendid illustration of a case in which the patient's epiglottis has been removed and in which men like Dr. Carmody and others have removed the epiglottis over and over again with benefit would indicate that we should not be so hasty in prognosis. If you will recall the picture showing infiltration in the posterior commissure, I think that will be a matter of great help in getting at the probable diagnosis very early in the case. Now it was stated that examinations should be careful and here again I want to emphasize what I said this morning, and that is, we must never be satisfied until we examine the parts to which we are accustomed. So when a patient has a cough his uvula, larynx and everything should be borne in mind. Attention was called in the paper to the need of having your iodine in bottles that would be free from evaporation. No drug that I know of changes in character like iodine.

DR. THOMAS E. CARMODY, Denver: I want to thank Dr. Dworetzky for his very thorough paper, and Dr. Mayer also, whose experience in this line we have known for years. As to prophylaxis Dr. Dworetzky covers that very carefully. They should be in the sanatorium or in the office by themselves. Give them a spray to use themselves and keep them in a very good condition. In the relation to lung diseases as far as we are concerned in Colorado I will have to agree with Dr. Dworetzky. We do not see the primary cases and most cases are sent West on account of pulmonary conditions. And all of our cases are pulmonary cases. As to rest and office treatment I believe that no patient should come to the office with an active lesion in the lung because you are simply working against that patient's welfare, and in those cases I give a spray to use at home three or four times a day. The spray we use is one-half per cent. formalin and Dobell's solution. As to epiglottidectomy, we have found very good results in many cases if the epiglottis is removed thoroughly. In the cases in which you have syphilis and tuberculosis they are unfavorable. When the pharynx and mouth become involved they are practically all hopeless. Once in a while you will find a lesion in the mouth that will heal.

DR. A. H. ANDREWS, Chicago: I want to speak about palliative treatment. The old orthoform treatment is still helping these patients and Dr. Carmody mentions formalin combined with Dobell's. The carbolic acid in the Dobell has an anesthetic effect on the larynx which is pleasing and if you add a little more carbolic to the Dobell they will use it more. The doctor mentioned two very important things in connection with the local and constitutional treatment. Get air and plenty of it. Get food and plenty of it and an appetite to take care of it. He did not mention a third, and that is, a good time for the patient. They then stand a much better show to get well than under opposite conditions. This is a little digression. If any man here has seen a case of tuberculosis acquire diphtheria I wish he would come and tell me of it for I am working something up on that line. I had one patient with tuberculosis get diphtheria and both conditions cleared up.

DR. W. LIKELY SIMPSON, Memphis, Tenn.: I want to emphasize one point. As has already been said, practically all these patients have tuberculosis of the lung. A good many of our best internists believe in rest in bed for these cases, and, if that is good treatment, certainly in any case that has tuberculosis of the larynx, as well as of the lung, he should be kept in bed. It seems to be poor treatment to have any of these patients with active tuberculosis of the larynx coming to our office.

DR. JULIUS DWORETZKY (closing discussion): I want to make clear the statement when I said that a lesion of the acute type and at the epiglottis usually takes a fatal course. This generally happens in the cases in which the disease originates at the epiglottis, but when the lesion begins elsewhere in the larynx, as for instance, in the posterior commissure and later involves the epiglottis, the prognosis is not necessarily grave. As to alcohol injections into the superior laryngeal nerve, I never had any personal experience with it, but I understand that other men have used it with good results. The relief of dysphagia following alcohol injections, of course, is only temporary.

CONSERVATISM IN RADICAL SURGERY OF NOSE AND THROAT

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The recent literature on focal infection and the excellent work done by such able investigators as Rosenow, Davis and others needs no comment but rather quite some time might be spent in words of praise and commendation for the very valuable statistics due to their untiring efforts and research.

Great as has been the relief to suffering humanity, as a result of the enucleation of the infected tonsil, yet there are a few of us who cannot but point to some patients in whom the satisfactory result was not obtained by such procedure. Statistics show that approximately 75 per cent. of all patients operated on were given total or partial relief from their symptoms. While this is a considerable majority, it proves that we have not yet reached the point of 100 per cent. proficiency.

It is seldom that a dissatisfied patient returns to inform us of his disappointment, yet he never fails to inform his fellows that a tonsillectomy did not cure him. Owing to this unfortunate state of affairs, and the knowledge of the laity in regard to the numerous tonsillectomies performed, I have been impressed with the idea that a more careful diagnosis should be made and a more guarded prognosis given in the obscure cases.

Permit me, at this time, to state that I have no sympathy for the old time tonsillotome and also no leaning toward some of the newer fads such as "circumcision" of the tonsil and "slitting the crypts." I do not wish to be classified as a "spray artist" or one opposed to surgical procedure, as my record of several years of practice will attest. I do countenance and practice complete enucleation of the tonsil by the Sluder or modified methods and dissection, my preference being dissection.

As a result of the popularity of the removal of the infected tonsil, the otolaryngologist is beginning to receive the same ridicule that the abdominal surgeon received a short time ago when he promiscuously removed the ovaries and later the vermiform appendix. No doubt but that some of this criticism is just, owing to the fact that some improper work is being done by the general practitioner or family physician who may be totally

unfit to perform such work or incapable of making a proper diagnosis.

Within the last five years we have come to recognize the terms "rheumatism" and "neuritis" as vague and indefinite maladies meaning anything or nothing. The bacteriologists have informed us that there are about 66 varieties of so-called "rheumatism" or "neuritis," and the moment we have given a patient a diagnosis of one or the other conditions we have unconsciously admitted our failure to make a definite diagnosis.

A short time after the faucial tonsils were popularized our sister profession came forth with the infected gums and teeth and then up came the abdominal surgeon with the infected gall-bladder. The so-called "rheumatic" or "neuritic" conditions we know to be due to infection and at the door of these three avenues of infection has been laid the causative factor for their production.

TONSILLAR INFECTIONS

Patients suffering from acute articular infections or the acute inflammatory conditions following immediately or shortly after an acute focal infection can be given almost absolute assurance against such recurrence on the enucleation of the tonsils. These, however, are not the patients who give us the most concern and for whom we are asked to advise some means of relief from their inconvenience and suffering. The subacute or chronic aching in joints, necks, arms, legs or backs in patients of 40 years of age or more, are brought to us for the purpose of determining whether or not the infection is focal in origin. These are the patients with whom we should be very conservative in regard to recommending tonsillectomy and then with a guarded prognosis.

It is my opinion that seldom, if ever, should the tonsils be removed, on suspicion that in them lies the focus of infection, unless some evidence of the presence of pus may exist, or a history of tonsillar infection be obtained.

We know of a very small percentage of patients who have been injured or impaired by tonsillectomy properly performed, yet there are on record cases of arrested tubercular lesions in the chest which "flared up" following tonsillectomy. One such case was observed by the author.

A man, aged about 45, a candidate for a national office, had a complete enucleation of the tonsils after which he spent about two years at a tuberculosis camp, the result of enlivening a

tuberculous area which had been apparently arrested for over twenty years.

The author also observed an infarct in another patient which started as follows: The patient, aged about 35, having had the tonsils enucleated, had pain in back over right lung the day following. No lesion of any kind could be discovered. Patient had a temperature of 99 F. Pain persisted for a week at which time she felt something "tear loose" or "break," and she expectorated a large quantity of pus. This condition continued for about four months and gradually grew less until after about one year's time she had completely recovered. She is now well and strong.

These are two cases in which a closer observation might have discouraged a tonsillectomy especially the first one, yet all of us can point to some unusual conditions which have apparently cleared up after operation.

OTHER TYPES OF CASES

Second only to the so-called "rheumatics" and "neuritics" in the source of discomfort or worry to the rhinologist, are the so-called "neuralgics." These patients suffer pain varying from a slight discomfort to one of a distracting nature which may place them in bed for one, two or three days.

The symptoms which these patients present vary to a very marked degree. Some complain of a continual inability to breathe properly through the nose and of "catching cold" easily. Others complain of a chronic discharge from the nose, while others complain of no discharge at all except a continual dropping into the throat. A few are not conscious of any nasal disturbance except it be a "drawn" or "tight" sensation just below the eyes.

The pain or discomfort which brings these patients to the rhinologist varies from one which they can locate definitely to one indefinitely located and terminating, they know not where. These pains may be constant or intermittent and may be located just above the eyes or just below and radiating outward over the lower border of the orbit. They may be located along the temporal bone radiating to the occipital bone or may be confined to the vertex of the skull. It is not an infrequent occurrence to have an indefinite pain covering the entire skull.

These conditions, as we all know, may be caused by several different factors such as infected ethmoids, frontal, maxillary, and sphenoidal sinuses and deviated septa with the coexisting middle turbinate pressure.

DIFFERENTIATION OF PAIN

Granting that fully 75 per cent. of head pains are nasal in origin, which percentage may be somewhat low, it nevertheless may entail some sharp differentiation between a referred pain and a localized pain. Eliminating the infected sinuses leaves us to deal with the deviated septal and middle turbinate conditions as causative factors which are often times much more difficult tasks than they would at first appear.

The neglected adenoids of childhood or injuries sustained during that period, have changed the true and ideal straight septa until they are seen about as infrequently as is a dispensary patient who has not had a Wassermann. These deviating septa all have some coexisting turbinate hypertrophy. It was just a few years back that the nares were mutilated by the electric cautery and later by the indiscriminate removal of turbinates. The gradual evolution in rhinology has brought us to the more sensible procedure—that of the submucous resection of the septum.

RESECTION OF THE SEPTUM

As has been stated, it is a very common occurrence to see a deviated septum and yet it is sometimes extremely difficult to determine to just what extent this deviation is an inconvenience to the patient and how much benefit will be derived from a resection in these border line conditions, except to arrive at a conclusion by the process of exclusion and careful observation.

Aside from the possibility of infection which now is somewhat remote, or the seldom but nevertheless occasional "saddle back" complicating the end-result of a resection of the septum, the dangers of such procedure are exceedingly few in number. However, the question of how much benefit has been derived may sometimes be a mooted one.

Among some cases which have come under my observation I wish to briefly mention four.

REPORT OF CASES

CASE 1.—Mrs. P., aged 60, complained of pain more or less constant above the right eye. The right middle turbinate was removed without much relief. The septum was resected and in less than six months after, the pain had never subsided. Alcohol injection of the supraorbital branch of fifth nerve relieved the symptoms.

CASE 2.—Mr. W., aged 50, complained of inability to breathe and profuse watery discharge for several months. The septum

was deviated and resection was advised. Three months after resection the condition returned. The antra were tapped with no result. Local treatment and vaccine have established a normal state.

CASE 3.—Miss G., aged 24, complained of a “stuffy” feeling in nose and inability to breathe properly. She also complained of some headache. The septum was somewhat deviated and resection was advised. No relief was afforded by the resection. The condition was entirely relieved by an abdominal section which corrected a pelvic condition.

CASE 4.—Mrs. M., aged 27, complained of pain below right orbit. The septum was deviated. A resection was performed, and later the antrum was opened intranasally. The roentgen ray revealed an impacted third molar tooth, the removal of which gave the patient her first relief from pain.

In conclusion, I would say that the good we do is sometimes greater because of our inactivity than by reason of our labor.

THE FUTURE OF PLASTIC SURGERY OF THE
FACE IN THE UNITED STATES. LANTERN
SLIDE DEMONSTRATION

JOSEPH C. BECK, M.D.

CHICAGO

In attempting to foretell the future of anything, one should consider the past and present, in order to be fair. It is not different with plastic surgery. I shall not attempt to dwell on plastic operations on other than that portion of the body situated above the clavicle, but I shall consider in addition some of the general principles of plastic work.

The word plastic refers to remaking or readapting organs and structures which have been destroyed by accident or disease, also those having congenital defects. The history of plastic surgery of the nose dates back several hundred years and some of the earliest work is illustrated by Togleocozzi. The gradual development of new modifications of the older methods is well shown by the gap in the literature of the seventeenth century. When the great Celsus brought out his new ideas of making plastic flaps, this work received a new impetus, and many cases were reported in which nose structures had been successfully reconstructed. The men who achieved success in rhinoplasty were, however, general surgeons and since there were no rhinological surgeons at that time, they became very proficient, first, because the field was fresh, and second because they had not so much other surgery to do, such as operations for appendicitis, gallbladder affections, etc. In the very recent past, even in the latter part of the last century, this work has continued to be performed by general surgeons, and excellent results in rhinoplastics were obtained by such men as Senn, Carl Beck, Syme and others. These men, however, became so busy with greater general surgical problems, that they permitted rhinoplastic surgery to fall in the background, as it were. They were not as enthusiastic and did fewer of these operations and consequently became less proficient especially on the cosmetic side of the question. Their attitude led naturally to the development of the quack and beauty doctors. These advertising quacks became so numerous as to take up a half page each of advertising space in the newspapers, and their obnoxious signs decorated

our main business streets. Only recently, since the reform propaganda of some of the newspapers, have they diminished in numbers, but this diminution is only apparent. They are doing business at the old stand by other means of advertising.

The advance in present day rhinoplasty should be accredited to an American rhinologist, who not only understood the physiology of the nose and the surgical and plastic principles, but who was also an artist and one of the first to designate the types of noses and faces and how they best agree from the artistic point of view. He was furthermore a great cosmetic master, and took a great pride in being able to rebuild a nose all from the inside, that is, not to make any external scars. Of course his work was principally on patients, in each of whom the nose structure was present, although markedly deformed. I refer to John Roe of Rochester. On the continent there was, or perhaps still is, a master of rhinoplasty, who has done a great deal to popularize the intranasal method of correcting deformities. But he, too, was most interested in cosmetic and not reconstructive work, by which I mean either building up a flat nose or, more often, taking off a hump, but not making a new nose. In other words, he was more on the order of a beauty doctor. I refer to Joseph of Berlin. Another American who has recently contributed considerable to the subject of correction of nasal deformities is Carter of New York, a rhinologist. His work has thus far been principally on the elevation of saddle noses, but from a recent article of his and from photographs he shows relating to his cases, I am confident that he will broaden into the field of reconstructive rhinoplasty. Still another rhinologist who has come into the arena of rhinoplastic surgeons is Cohn of Baltimore, of whom I am expecting great things in the future of rhinoplasty.

My hobby has always been the correction of nasal and ear deformities, and long before I took the special field of rhinology, laryngology, and otology, I had the opportunity to see external plastics of nose and ear, and to assist with the operation and to do them myself. It is to my brother, Dr. Carl Beck, and to Drs. Nicholas Senn and Truman W. Brophy that I desire to express my gratitude for having taught me much of what I know about plastics of the upper region of the body.

In 1906 at the annual meeting of the American Laryngological, Rhinological and Otological Association, I presented my first comprehensive work in the form of case reports from my experiences during the period since 1895. In 1908 I presented

before the Chicago Laryngological Society a number of cases which had been corrected by rhinologic and otologic plastics. Since that time, I have repeatedly presented before the same society cases of the same type showing what could be done in the reconstruction work.

In 1916, I collaborated with Dr. H. W. Loeb, by writing the chapter on "Plastic Surgery of Nose and Ear" in his textbook on surgery of ear, nose and throat. I mention these facts to express my surprise that more men have not been encouraged to take up this most interesting work and report on their results, because it has been my aim to illustrate my case reports profusely and in the textbook I described the operations step by step so as to stimulate men to do the work. I have heard it expressed by men visiting my clinics, that they have attempted to learn this work by going to the men reputed to be able to teach them, but they never were able to see these men operate. This complaint was lodged particularly against physicians on the continent. It has always been my practice and pleasure to illustrate to the visitors each important point in the technic, and I believe it is the duty of everybody to do likewise. This is because these operations can best be learned by that method. How much better off the United States would be if more men were able today to do this work, which is so important in view of the conflict in which our soldiers are being wounded, and their faces distorted. These men, unless their deformities are corrected or at least improved by plastic surgery, will be or will feel themselves outcasts and curiosities, and so become burdens to society and to themselves.

Furthermore, there is a much larger proportion of people in the United States in civil life than most of us realize that has true extreme nasal and ear defects which should be corrected and are not. Why not? I have made the observation from a fairly large mass of material that most of these defectives come from very humble walks of life, and their usual answer to my question as to why they had not been operated on before was that they were not financially able. Is it possible that our noble profession could be accused of not being willing to do for these poor people what is right? I am sure that that accusation can not be substantiated. More often than members of other professions does the physician take care of the poor and more often is he taken advantage of. Then what is the reason? My belief is that most men lack information on this subject, but I trust that this condition will soon be remedied, for I fear this knowl-

edge will soon be greatly needed. Surely there will be plenty of opportunities for its use after this war. The number of men of our Allies as well as enemies who have been hit about the head in this trench war is enormous in comparison to previous wars, and the defects in the external nose and ears caused thereby have not received immediate cosmetic attention by specialists. They are usually treated in such a way as to obtain as rapid healing as possible, so that the men may be returned to the front as soon as possible. This has been the first duty of the military medical man. Although I have no authority to speak of the intentions of the surgical heads of the United States Army, it is my belief that in their organizations they intend to pay more attention to the immediate physico-physiologic and cosmetic correction of their wounded. It is known to all physicians, I believe, that the Surgeon-General has ordered the establishment of a head hospital where the affections of the head are treated by specialists, each working according to his recognized special attainment. Plastic and oral surgery is one of the subdivisions. Major Blair intends to create schools for the education of military medical men, who have been assigned to the plastic and oral surgery department before they are sent abroad. These schools are to be located in some of our larger cities, in the different sections of the country.

I will now present in review some of the cases of defective or deformed noses and ears, in which I have performed operations, simply calling attention to the type of deformity to which each belongs. I shall not describe any technic nor show the end-results except in one or two instances. I will say, however, that the best results obtained were far from ideal, and from anything approaching the normal appearance one would like them to have.

At the same time, there is not a more appreciative and grateful patient than one who has had a marked deformity that has been improved, even if only partially. It is entirely different in the case of that type of person who comes to the plastic surgeon because he doesn't like the shape of his nose—the so-called hypersensitive deluded self-centered, neurotic, foolish, vain man or woman. This class, of course, is not to be considered in connection with the subject in question. I believe, however, that it is our duty to take care of such patients, so as not to allow them to fall into the hands of the quack, who will fleece them of their money and oftentimes cause an irremedial damage. These patients, in contradistinction to those having real external nasal

and ear deformities, belong to the wealthier class, and I am sorry to note that their cases make up the greater number of reported cases in rhinology and otology. I have said nothing about paraffin, because its use is practically extinct in the practice of rhinoplastic surgery, though remaining the greater armamentarium of the faker.

DISCUSSION

DR. HARRY L. POLLOCK, Chicago: The subject presented by Dr. Beck goes to show some of the work we have been doing. There is certainly more need for plastic work and a better understanding of the principles. It is a branch of our work entirely different from the main part. Certain principles must be understood before we can expect any degree of success. Unfortunately most of these cases are among the poorer class of people, that is to say, a great many of our depressed noses are due to syphilis and these cases are neglected. One thing Dr. Beck did not state is that in practically all the nasal deformities it is necessary to build a support. Years ago, most of this work was done by injections of paraffin. We have practically discontinued this practice, because either the paraffin sloughs out or causes a sloughing of the skin, and the resulting deformity is greater than the original. It is necessary to use a bony frame work. To get a good support it is necessary for the transplant to come into contact with denuded bone. We do intranasally most of the work, in fact all of the work, in which previous operation has not been done, or in which there is no external scar. If there is a scar on the nose, the work can be done from the outside. The most remarkable thing I see in this work is the small amount of infection that takes place. Of course, it is absolutely necessary to be as sterile as possible, but when one goes through the nose where one cannot completely disinfect the membrane, very few infections result. It is just recently that we have been using cartilage. Heretofore, it has been thought that cartilage would be absorbed with or without its perichondrium. It is much easier to adjust the cartilage than the bone. We have also found that far better results are obtained if we take several particles of bone. One of the main things in plastic surgery is to get enough loose tissue, so that there will be no tension, and I know some of our earlier cases were not successful because we failed to observe this rule. The sutures will tear out and the resulting scar will be as bad as before the operation.

DR. JOSEPH C. BECK (closing discussion): I do not think I have anything further to add except the point which Dr. Pollock brought out. This question of cartilage transplants is interesting. You find that Quain and J. B. Murphy in his early work said that cartilage, either from disease or trauma, when once severed from the perichondrium, would be absorbed, and Zaufal in his work on "Hematoma of the Septum" stated it would always be followed by a sinking-in of the nose because the hemorrhage lifted the perichondrium on both sides and the cartilage died, even if the clot is emptied immediately. I have until very recently never used cartilage for building up the nose or ear for this reason. The physician of whom Dr. Pollock speaks, and in whom the late Dr. Beckman of Rochester, Minn., implanted a rib for a sunken nose, came to me saying that his nose was now too large. He had a typical hump nose. That operation had been done several years back and he said it was cartilage that Beckman implanted. So after operating on him and removing the excess of the lump, I took a section to the laboratory and it showed a typical cartilage of no active growth with its perichondrium.

Consequently it is advised that perichondrium should not be used in transplanting the costal cartilage. Last week Dr. Grant of Denver informed me he had done several cases of costal cartilage and he vouches for the fact that they remained alive. If that is true, we have solved the problem of curing alar collapse. The use of cartilage will also solve the problem of shaping the ear. Cartilage is very easily obtained as f. i. from submucous resections. One more point that is very important is the blood supply. One thing to know is the anatomy and course of the supplying vessels to the pedicle of flaps, otherwise you will reduce the chances of the flap you are making. We have had cases of congenital or hereditary syphilis in which not only the cartilage but the entire nose was destroyed.

DISEASES AND DEFORMITIES OF THE NOSE VERSUS NEURALGIA OF THE HEAD

FREDERICK STAUFFER, M.D., F.A.C.S.

SALT LAKE CITY

I have long been impressed with the importance of a more restricted use of many words denoting symptoms, which through common usage have assumed the rôle of disease classification.

Among these, there is perhaps no word which has been more misused than that of "neuralgia."

We all know how common is the practice of diagnosing all kinds of diseases about the head which cause pain, as neuralgia, and prescribing some favorite analgesic to relieve the symptoms without due regard for diagnosing the underlying cause and directing the remedies at the disease instead of attacking the symptoms.

Though my purpose may not be well expressed in the title of this paper, I shall endeavor to point out the fact, that when all the head pains or neuralgias occurring as a result of disease and deformities in the nose and its accessory sinuses are accounted for, there will be but few to attribute to other causes.

DEFINITION

Gould's definition of neuralgia is: "Severe paroxysmal pain along the course of a nerve and not associated with demonstrable structural changes in the nerve."

The word has been used also to indicate conditions in which such pain exists as a pure neurosis, to distinguish it from the cases in which inflammatory and degenerative changes are present in the nerve, to which class the name of neuritis is applied.

Pain may be regarded as a reaction of the organism, in part or whole, to harmful influences, giving a warning in consciousness that some activity prejudicial to the health of the tissues is present.

Biologists say that the movements of expansion and contraction of protoplasm are primordial expressions of the pleasure and pain sense; expanding to pleasure giving, and contracting to pain giving or harmful impulses.

Hence, any agency in the system which through pressure,

chemical or other irritation, causes undue contraction of the nerve protoplasm tends to cause pain.

Sensations of pain are conveyed to the brain centers through the afferent, or sensory nerves, and the pain is referred to the origin of the nerve fiber whether the irritant is applied to the periphery or to any part of the nerve in its course to the brain.

ANATOMIC REVIEW

The trigeminus is the great sensory nerve of the head and face, and through its ramifications are conveyed to the brain all manifestations of common sensation in health; and the sensations of pain, expressed in neuralgia, when its protoplasmic elements are disturbed by disease of the surrounding tissues.

A knowledge of the origin and distribution of this nerve is imperative for a proper understanding of the various obscure and reflex pains associated with disease in the nose. However, a minute anatomic description of its origin and ramifications is not called for at this time.

Irritation of any branch of this widely distributed sensory nerve may cause pain referred to any other branch by reflex irritation. While the *modus operandi* of this phenomenon is not perfectly clear, there is a more or less uniform symptomatology from certain specific causes which bring about these reflexes.

Referring again to the definition of neuralgia, it is difficult to conceive of pain in the course of a nerve as a pure neurosis (whatever that means), without a lesion either in the nerve endings or nerve fibers in their course to the sensorium.

Taking as a basis for discussion the hypothesis that pain sensations are caused by influences which cause contraction of the protoplasm, we may account for many neuralgias, where no demonstrable lesion of the nerves is present.

ETIOLOGY

Some of the predisposing causes of neuralgia are: anemia which acts by impoverishing the nutrient fluid and overcharging it with acid products, the presence in the blood of abnormal substance resulting from imperfect metabolism; also such diseases as rheumatism, syphilis, arteriosclerosis and all other conditions which interfere with proper nutrition of the nerves, may of themselves cause neuralgia.

To the rhinologist, however, it is more important to discuss the subject from the standpoint of peripheral irritation, especially

the irritation which comes from pressure in deformities in the nasal cavities and from pent up pus in its accessory sinuses.

SYMPTOMS

Since pain is the leading symptom for which patients consult the doctor in all of these conditions, I can best serve my purpose by describing the character and location of the pain which, according to my observations, are fairly reliable diagnostic aids.

The pain in acute frontal sinusitis or in the acute exacerbation of chronic inflammation is described as dull, heavy, throbbing in character and is intensified by body movements which tend to increase congestion, such as stooping forward; or in jarring the body as by running or jumping. The pain is usually limited to the region of the sinus itself and is greatly increased by pressure over the floor of the sinus at the upper inner angle of the orbit.

One of the most characteristic symptoms of frontal sinus pain, is its periodicity, starting usually between 9 and 11 a. m., reaching its greatest intensity early in the afternoon and disappearing before 6 p. m., without treatment, only to recur on each succeeding day, until the pathology is relieved by spontaneous evacuation of the pus, or by the aid of the physician.

The pain is often as intense in the congestive stage of an acute frontal sinusitis before pus has formed as it is in the later stages when it is ascribed to back pressure of pus. In the former case, the pain is probably due to congestion of the mucosa because of a vacuum in the sinus resulting from closing of the infundibulum and absorption of the contained air. In any event, the pain is often relieved and suppuration aborted by applying astringents to the nasal mucosa at the infundibulum whereby the air passage is reestablished.

As before observed, the pain in frontal sinusitis is usually located directly in the region of the sinus, and no reflex pains to other parts of the head; hence the diagnosis of this disease from the pain alone is easily made. The same may be said to a limited degree of maxillary sinusitis; there is usually a dull heavy boring pain below the orbit which may or may not be increased on pressure over the canine fossa.

While the pain is usually most pronounced over the antrum, it may be entirely absent and be referred to the supraorbital region or along the backward extension of the superior maxillary nerve, which supplies the antrum.

Nevertheless, the pain in maxillary sinusitis is not often mis-

leading nor wholly referred to distant parts. In this respect the sphenoidal sinuses and posterior ethmoidal cells differ materially from the frontal and maxillary sinuses.

Careful observation and study of pain arising from irritation in the sphenoidal and posterior ethmoidal sinuses has led me to look for and find pathology in these cavities when there was not the slightest suspicion on the part of the patient or his family physician of the location of the disease.

While the pain may be located in the region of these sinuses, it is more often referred to the parietal or occipital regions.

Every rhinologist can recall patients who applied for ear treatment because of pain referred to the middle ear or to the mastoid bone, when on examination the ears were found perfectly normal and the lesion was found in a diseased sphenoidal or posterior ethmoidal sinus.

The pain from pent up pus in the sphenoidal sinuses and posterior ethmoidal cells is often intense and throbbing or boring in character. It is nearly always referred, either to the occipital, mastoid or parietal region of the same side as the involved sinus, and in contradistinction to the frontal sinus pain, it is usually severer at night.

I report the following case as typical of a class to which I wish to draw special attention because of the great frequency with which the real lesion is overlooked and the symptoms attributed to other causes.

REPORT OF CASE

Mrs. J. E. S., aged 30, mother of three healthy children, came for treatment with a history of having had severe neuralgia in the left side of the head radiating from the base of the middle fossa up to the occipital, parietal and frontal regions of the head, for a period of three years, with only occasional remissions. This pain had been so severe and long continued that the patient, as well as her husband, feared insanity would be the end-result.

She had been to numerous opticians and oculists who prescribed various lenses and ran through the category of analgesics with only temporary relief of pain. This patient denied having any trouble in her nose, but was convinced that the cause of her trouble was in her eyes, if not her eyes then there must be a lesion in the brain.

On first examination, Dec. 5, 1914, I found an error of refraction which was corrected by $+75$ sph. in right and $+50$ sph. with $+37$ cyl. ax90 in left. As the patient gave a negative history, I made no examination of nose at this time, but finding this error of refraction, contented myself with prescribing the

proper correcting lenses in the hope that these would bring the desired relief.

Feb. 16, 1915, patient returned reporting that headaches still continued as before. At this time careful examination of the nose was made and nasal septum was found to be deflected to the left, high up, causing considerable pressure on left middle turbinal and its impaction was so great that it wholly obstructed the opening of the sphenoidal sinus. Closer examination revealed some pus in the region of the left sphenoidal sinus opening.

I made a submucous resection of the nasal septum, removed the impacted middle turbinal, and enlarged the opening of sphenoidal sinus; this procedure gave prompt relief from the pain for several months.

Oct. 23, 1915, patient came back completely discouraged, now having pain on both sides of the head.

Up to this time she had never had pain on right side; but now the patient was suffering from an acute coryza. By shrinking the nasal mucosa, it could be seen that the right middle turbinal which was hypertrophied before the submucous resection was made, had become impacted as a result of moving the septum toward the median line, and there was an acute suppuration in the right sphenoidal sinus as well as an acute exacerbation of the left sphenoidal sinusitis, which condition caused the pain in both sides of the head. The right middle turbinal and also the whole of the anterior wall of both sphenoidal sinuses were removed. After a period of treatments extending over several weeks, the pus from both sinuses cleared away and, with reestablishment of normal ventilation in these cavities, the pains in the head entirely disappeared and the patient has been well and happy since.

COMMENT

It is because of the large number of cases of this character which have come under my observation, that I am led to believe, that a very large majority of all these obscure neuralgias (so called) in the head have their origin in diseases of the accessory sinuses. Exostoses or deflection of the nasal septum, which cause pressure in the region of the posterior ethmoidal cells often cause similar symptoms. Undue pressure in the region of the anterior ethmoidal cells causes pain between the orbits and in the vertex of the cranium.

The neuralgias arising from disease of the Gasserian ganglion, as expressed in "tic douloureux" and supraorbital neuralgias, as well as those due to carious teeth, I shall not attempt to discuss, as they are usually easily differentiated from those under discussion. The diagnosis of diseases of the accessory sinuses is not always easy and can be made only by careful study

of the clinical symptoms and physical signs. In recent years the roentgen ray has been extensively used in diagnosis and, while it gives valuable diagnostic information, I am convinced that many cases of well advanced disease of these cavities have been overlooked through laying too much stress upon the roentgen-ray findings.

I am more than ever convinced that the court of last resort in diagnoses of accessory sinus disease, must be a careful study of clinical symptoms and physical signs.

Every case with obscure head pains, should be subjected to a critical examination of the nasal cavities before and after blanching their mucosae.

By this procedure it is easy to ascertain the presence or absence of the most essential elements of a healthy nose, namely, ventilation and drainage.

In the absence of either of these conditions there cannot be a normal functioning in this important organ.

TREATMENT

The treatment, therefore, may be summed up in two words, namely *ventilation* and *drainage*. These conditions being present there cannot be pain or neuralgia such as I have described.

It is the absence of them that causes untold numbers of victims to exhaust the category of so-called neuralgia and headache cures with only temporary or no relief.

DISCUSSION

DR. G. C. KNEEDLER, Pittsburgh: The subject of pain as chosen by Dr. Stauffer is always appropriate, for there is no symptom in the category of medicine which brings a patient more readily to the physician than that of pain; nor is there anything that we do which wins greater appreciation of our services, than the cure or relief of same.

As to cause, headaches may be classified as local and general. The local headaches associated with sinus infection are extremely common. Hajek says "many cases of sinus disease with slight nasal symptoms go through their entire life with the diagnosis of chronic headache, taking all manner of cures such as electric and hydrotherapy, sea-baths, general and special baths, without it occurring to any one that the headaches might be caused by a structural disease in the immediate neighborhood, namely, that of the accessory sinuses of the nose. It is barely possible that some of these patients have not had a thorough rhinoscopic examination and are consequently overlooked. If Samson had not been lying around all day with his head in Delilah's lap he would not have been shorn of his power. So with many examinations—they are made without due care and a lack of thoroughness; therefore results are not secured because the *cause* is not recognized.

We do not always find a profuse discharge from the sinuses, neither is it always fetid. In many cases the mucosa shows on the surface few

pathologic changes, especially is this so when the secretion thrown out is scanty or of a thin, serous nature. The cells may be filled with hyperplastic or cystic mucous membrane of a degenerative character, and very little discharge shown. When pus is seen to flow from the cells, a diagnosis of some pathology is easily made. In most acute inflammatory conditions of the mucous membrane, the surface may be covered with a thick creamy pus; but if this is carefully removed and returns immediately or is shown again with the use of some suction apparatus, we may be sure that the pus is coming from the cells beneath, rather than that of the mucosa per se. Under certain conditions a purulent sinus discharge will cause the formation of polypi, and in others very little discernible change takes place in the mucous membrane, except hyperplasia or atrophy of same. Besides the direct illumination of the nares, every means at hand for a proper diagnosis should be used, including transillumination, the roentgen ray, the nasopharyngoscope, and if need be, the puncture of the cells.

Deformities of the septum such as deviations and spurs, especially when they impinge on the turbinates, or obstruct in any way the normal drainage from the nasal sinuses, or a unilateral breathing space, may be looked on as a causative factor when existing. The deviation high up is the one most likely to be overlooked in our rhinoscopic examinations. I have found the correction of these deformities by a submucous resection to give great relief when drainage and aeration of the attic is secured. The mere fact of pressure on the swollen turbinates may cause excruciating headache. This type of neuralgia may be relieved by the application of cocaine and adrenalin to the swollen mucosa and will sometimes help us in making a diagnosis of pressure pains.

Causes of neuralgias of this type may be classified as follows: (1) obstruction to normal drainage, thus causing reabsorption of toxins within the sinuses; (2) interference with cranial circulation; (3) congestion of the mucosa; (4) disturbances of the lymphatic and blood circulation near the base of the skull; (5) overindulgence in alcohol, tobacco and excessive eating may be causative factors in the production of neuralgia of the head.

The correction of these conditions resolves itself into two things, namely, that of a thorough rhinoscopic examination for the proper diagnosis and the removal of the *cause*, which can nearly always be considered a surgical procedure. The restoration of the function of the nose by restoring the normal air current, relieving pressure, securing drainage and aerating the cells is always the procedure to follow. The general causes of headache should be left to the internist.

DR. G. B. JOHNSON, Franklin, Pa.: Dr. Stauffer's paper has been of considerable interest to me, as it coincides with some statements which I made, in a paper on "Trifacial Neuralgia from Nasal and Accessory Sinus Disease," read at a meeting of the Pennsylvania State Medical Society, in 1914.

The discussion of this paper showed a diversity of opinion, as to the etiology and pathology of trifacial neuralgia, and some skepticism as to a cure by nasal route. I gave clinical histories of a number of cases which were cured by remedying sinus abnormalities and intranasal growths. Since reading this paper, I have successfully treated other cases of trifacial neuralgia, intranasally.

This, to my mind, is sufficient evidence of the existence of cases of trifacial neuralgia, due to peripheral irritation.

My first successful patient had consulted successively internists, neurologists, ophthalmologists, and a dental surgeon, without gaining relief.

An intranasal examination of this patient revealed nothing which was apparently abnormal. However, after spraying the nostril on the affected side with a weak solution of cocaine and adrenalin, the pain ceased temporarily. The lower turbinate was pushed aside, and a large spur was encountered, the apex of which was embedded in the extreme posterior end of the lower turbinate. The spur was removed, and eventually the severe trifacial spasms ceased. The cure of this patient brought others for relief, many of which were cured, and some were not. The latter were dependent on constitutional states of the body.

I do not agree with the opinion of many writers, that only a small number of trifacial neuralgia cases are peripheral in character; but if a more thorough examination by the roentgen ray and other means, were instituted, the vast majority would be classed as being due to peripheral irritation.

DR. WILLIAM A. MANN, Chicago: I want to emphasize one point and that is let the patients tell their tale and get a clear history of the case because that will very often go a long ways toward locating the trouble, for instance, a pain coming on in the morning you may refer very largely to the nose as the seat of trouble. A pain coming on in the afternoon you may refer very largely to the eye as the seat of trouble, especially if it is frontal and by far the larger percentage of these pains or aches are neuralgic as you call them or attributal to the eye. Then a certain number of them are caused by reflexes in some other part of the body. While a certain percentage, I would say 10 or 15 per cent. of these patients can be relieved, as Dr. Stauffer has said more than 60 per cent. are due to the eye and the remainder to some other part of the body.

DR. HAROLD BAILEY, Springfield, Mo.: I am glad the essayist defined the word "neuralgia." The term and its meaning are familiar to the physician but it is too often misunderstood by the patient. In our locality we see every year a number of cases of sinus infections and other chronic diseases that have been endured for years without treatment and without care, all because they were supposed to have "neuralgia" for which they thought nothing could be done. We have learned to regard the word as used by the laity as a very indefinite and ambiguous term. There is one condition which we occasionally see, though not often, in which the pain is due to the pressure between the superior turbinal and the septum. The pain in these cases is fairly constant and often extremely annoying. It is intensified during attacks of acute rhinitis. Relief follows removal of the offending turbinal. It has been my observation that this condition is frequently overlooked.

DR. B. F. ANDREWS, Chicago: This paper has been of great interest to me. I have been studying the fifth nerve for a number of years, and have gotten to be quite chummy with it. Some two years ago I read a paper before the Pan-American Medical Congress in San Francisco dealing with some reflexes of intranasal origin, and in it I referred to headaches and incidentally to asthma and other conditions. I am inclined to believe that, while there may be pressure from within the sinuses, most of the pressure irritation comes from malpositions and malformations of the intranasal structures normal to the nose, that is, the septum and the turbinates. Nothing has been said of pressure on the lateral wall and the floor by the turbinates. I have seen cases of pressure by the middle turbinal on the bulla ethmoidalis, corrected by fracture of the middle turbinate, thus letting it hang free from lateral and septal contact. The headaches have disappeared following this procedure.

DR. A. H. ANDREWS, Chicago: I like this term neuralgia. I confess it covers up our ignorance but we have lots of ignorance that needs cov-

ering up. When we find the cause it is not neuralgia. If it cannot be found, it must be neuralgia, and they all know how hard neuralgia is to cure. Just how these referred pains come about is interesting. I am convinced that the multipolar cells in the cortex are the explanation. The first branch of each of the great divisions is a meningeal branch, and the filaments from these branches may terminate in and arborize around the pole of these multipolar nerve cells, and one afferent filament from the nerve carries the impression up to the centers of consciousness. For this reason the centers of consciousness have no reason to know where the pain is located. It may be in the eye or the head or the ethmoid or the sphenoids, or the antrum. It may be referred to some part distant from these nerve cells. A greater familiarity with the fifth nerve taking under consideration these multipolar nerve cells will help us to understand some of these pains and encourage us to search further for the cause of them.

DR. STAUFFER (closing discussion): I want to emphasize the points made in the paper with reference to these pains. The point I wish to make is that pain is due to pressure. We may have predisposing causes but the main cause is pressure. Back pressure of pus, I think, is not so often the cause of pain as pressure from deformities. You cannot have proper ventilation and have pressure from deformities, and of course the pressure is the main cause of the pain.

HERPES ZOSTER OTICUS. A REVIEW OF THE LITERATURE AND REPORT OF A TYPICAL CASE

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INTRODUCTION

In 1835, there appeared in medical literature the first good description of herpes zoster. Preceding this there were, however, comments on the disease which date back to Hippocratic times. Known also as zona, shingles, radiculo-ganglionic syndrome and acute posterior poliomyelitis, the disease is an example of a cutaneous disorder which has a nervous origin, namely, inflammation or thrombosis of the posterior root ganglion. In the broadest sense it may be due to an acute or chronic meningitis, tabes, Pott's disease, carcinoma of the vertebrae, acute infectious diseases, intoxications or other lesions implicating the posterior roots and the ganglion, constituting a symptomatic zoster. In a narrower sense, and the one in which we are to view it in this discussion, it may be considered to be a specific infectious disease affecting the ganglion cells in the posterior spinal ganglia and nearby fibrillary structures, that is, essential zoster or posterior poliomyelitis. In the disease a crop of vesicles appear, distributed over a definite metameric area, that of the nerve distribution corresponding to the posterior root whose ganglion is diseased. In its manifestations this form of the disease is unilateral. In facial herpes it is the Gasserian ganglion that is affected. Herpes zoster ophthalmicus is a manifestation of this form where the naso-ciliary branch is involved in the acute descending neuritis following the ganglionic infection. This disease has long been recognized in ophthalmology and is frequently enough seen to be familiar to the ophthalmic clinician.

DEFINITION

The term herpes zoster oticus was first used by Koerner in 1904 and Gradenigo in 1907, being applied to the symptom complex dependent on involvement of the geniculate (seventh nerve) ganglion by this disease. Gradually our knowledge of the pathology of these clinical manifestations increased, until

in 1907 J. Ramsay Hunt, neurologist, published his monograph which, with subsequent ones, brought the disease to a definite ground in pathology and symptomatology.

ETIOLOGY

This is still a ground for debate. As in many other fields of medicine, when there has been a clearly defined analysis of what has hitherto been an imperfectly understood clinical picture, there have appeared many case reports and analyses until now there is a good literature on this subject, though as yet textbooks give but meager references to it. Besides the publications of J. Ramsay Hunt, one should not fail to mention that of Dr. Virginius Dabney which appeared in the *New York Medical Journal*, February, 1914, and that of Head and Campbell in the pathology of Herpes.

Naturally, a critical discussion of herpes zoster oticus, auris, or auricularis, implies a study of the general subject of herpes zoster. The author wishes to state that what appears herein is the result of studies of, and compilations from the literature, reference to which is given, and that nothing herein can be stated to be the result of original investigation beyond the mere case report which is a part of the paper.

As already stated, the disease is unilateral. There seems to be no reason why multiple involvement of the ganglia may not occur. Perhaps in the majority of cases only a single nerve root ganglion is affected, that is, if the Gasserian is involved, the geniculate escapes; if the geniculate is involved, others escape. Cases with plural ganglion infection are to be met with, and in the most pronounced, that is, severe types of any ganglion herpetic infection, the symptoms usually indicate multiple involvement. Hunt says, "The Gasserian, geniculate and upper cervical ganglia may be regarded anatomically as representing a more or less continuous ganglionic chain," and argues that "if the chief inflammatory or eruptive focus were situated in any one, a milder *noneruptive* inflammatory reaction might occur in other ganglia of the group."

Dabney states that "the modern consensus of opinion and the conclusion of most competent observers are that herpes zoster is an acute infection and while (quoting from Hunt) 'the specific infectious agent is unknown, it seems to possess special affinity for such ganglionic structures of which the posterior root ganglia of the spinal nerves and the Gasserian ganglion are types.' Loudouzy states that herpes zoster is a determination

on the nervous system of a general disease having for its apparent, that is, visible manifestations a vesicular eruption.

Medical literature seems interested in the question of the etiology of this disease. European references to it give chills and chilling as the usual cause and Dench mentions this, as the etiologic basis. Hammerschlag gives a rheumatismal cause, whatever that may be. Dietetic errors are mentioned by Dench; traumatism by another. Bacteriologic activities in the throat, or adjacent air passages, by production of toxins may be considered a possible cause.

There seems to be a desire to establish proof of its infectious nature. Thus a patient in Paris visited a small town where there were no cases, but in the six weeks following seven cases occurred there (?). A medical student is said to have developed the disease following the examination of a patient suffering from the same. Hay's views are quoted to the effect that (1) herpes zoster may be sporadic; (2) endemic in certain regions, for instance in California valleys as distinguished from the mountain region; (3) the course is similar to that of infectious exanthemata; (4) usually, but not always, there is immunity after one attack; (5) succession of cases in one family; and (6) quoting Graham's fatal case in which necropsy revealed softening of the brain, inflammation of the Gasserian ganglion, and around the fifth nerve leptothrix threads and bacteria were found in the arterioles of these structures."

PATHOLOGY

In studying the pathology of the disease, the posterior root ganglions show hemorrhagic changes and inflammatory foci which support, according to Osler, the theory of the infectious nature of the disease. At times, destruction of some of the single cells is found. Using this pathologic finding as the basis of a classification, Hunt distinguishes herpes oticus; herpes pharyngis, and herpes linguae and facial palsy; herpes of the cephalic extremity with facial palsy and auditory symptoms, and showing the presence of pain, paralysis, vesiculation, and deafness in proportion to, and limited to the nerve supply of the affected ganglion and its peripheral ending.

Head and Campbell in their noteworthy investigations of this disease made twenty-one necropsies in subjects who had shown a herpetic eruption of from a few days to one and a half years before death. They attempted to define the central areas represented by the skin areas affected.

Summing up the pathology, we may therefore say there is an acute hemorrhagic inflammation of the sensory ganglions. This causes swelling and thickening of the capsule and their infiltration with leukocytes. The inflammation is usually found in only a few ganglions. However, secondary degenerations may be found in the spinal cord and even an extension of the process to the cord. While we have distinguished between symptomatic and essential herpes zoster, in some of the cases of the latter type ganglia seem to have been unaffected, the only lesion found being a neuritis. There may be a lymphocystosis of the cerebral spinal fluid in the infectious types.

ANATOMY

A good understanding of this disease is to be reached only by a knowledge of its anatomy. The seventh or facial nerve is the most frequently paralyzed of all the peripheral nerves in the body, (cranial or spinal); and, as the implication of its sensory ganglion is the essential feature of this disease we should know its anatomic course and distribution. Like the fifth, it is a mixed nerve, with its motor root the facial nerve proper, and a sensory root—the *nervus intermedius* of Wrisberg—the two roots meet at the geniculate ganglion.

We may divide the nerve into three parts: (1) from its origin in the lower part of the pons to the geniculate ganglion; (2) from the geniculate ganglion to its exit at the stylomastoid foramen; and this again into subdivisions of (a) tympanic, and (b) mastoid portions; (3) that part lying external to the stylomastoid foramen.

The nerve supplies through its motor fibers all the mimic muscles from the occipitalis to the platysma. In the lower mastoid portion of the nerve is given off the chorda tympani branch which carries sensory fibers picked up at the geniculate ganglion from the nerve of Wrisberg. These ascend along the inner wall of the tympanic cavity, external to the incus but medial to the malleus, anastomosing with Jacobson's nerve from the glossopharyngeal. It drops and leaves the cavity by the Gasserian fissure, giving off branches to the otic ganglion, and goes medial to the ramus of the mandible, joining the lingual nerve of the fifth and thence to the submaxillary ganglion and gland.

After its exit from the stylomastoid foramen, the main stem of the facial picks up sensory branches from the fifth, explaining the burning and pain in those lesions of the seventh external

to the stylomastoid foramen. The geniculate ganglion is found in that portion of the nerve just after it has left the internal auditory meatus and winding along the aqueduct of Fallopius. Into it enter the sensory roots from the nerve of Wrisberg and fibers from the superficial great petrosal nerve from Meckel's ganglion and the small superficial petrosal from the otic ganglion. The geniculate is similar in structure to a posterior root ganglion and is sensory in function. Its cells are of the unipolar type and it is therefore the homologue of the Gasserian and spinal ganglions.

Inflammation of this ganglion will always be accompanied by herpes of the external auditory canal and adjacent part of the auricle. The facial nerve is therefore largely motor, though the geniculate is a sensory ganglion. The secretory and taste fibers, found in the chorda tympani, represent its sensory elements, though it is probable that the gustatory function of the nerve may be better explained by fibers from the glosso-pharyngeus than from the fibers of Wrisberg.

SYMPTOMS

Certain prodromata are to be expected before the full evolution of this disease. Localized hyperesthesia, which develops into a pain, may precede the eruption by a day or two, and is known as the preherpetic pain. Aside from this, indefinitely may be recognized such symptoms as indigestion and, rarely, fever. As to the symptoms themselves, pain is the most constant and distressing manifestation. This is deep-seated and, variously radiating, pulsating, lancinating, boring, or prostrating. Impairment of hearing is to be expected. More commonly this is due to the inflammatory swelling of the integument of the canal of the ear and partial closure of the same, but in the later or fully developed states of the disease this may be due to involvement of the auditory nerve branches. Dabney states that deafness is not to be expected as an early symptom, though in his case it was the first. The degree of loss of hearing varies. Tinnitus may be present without any deafness. The eruption appears after the disease has been established from two to five days and its classical distribution is in the external auditory meatus, and to a limited extent, the interior surface of the concha. These represent the cutaneous distribution of the geniculate. Exceptionally vesicles may be found on the membrana tympani, and a little more frequently on the auricle and over the mastoid region. Paralysis of the facial nerve may be

expected in the well developed involvement of the geniculate. This goes on to involvement of all the muscles supplied by the motor branches, the orbicularis being the one most apt to escape. There *may* be some paralysis of the palatal branches and impairment of taste though the chorda tympani involvement is usual.

While cases have been noted in which the attack was ushered in by fever, nausea, vomiting, and the full train of Ménière's symptoms, yet the common evolution is for the labyrinthine involvement to manifest itself after the symptoms already detailed have been in evidence. This involvement of the auditory from its proximity to the geniculate ganglion and seventh nerve is manifested by a more intense tinnitus and deafness, the latter increasing and becoming associated with, first, nystagmus, then vertigo, and, in the most marked cases, nausea, vomiting and disturbance of equilibrium. Where other ganglions than the geniculate have been involved there may be in addition pain and eruption on the side of the neck and occipital region, over the mastoid, laryngeal disturbance from vagus involvement; fifth nerve neuralgias when the Gasserian is affected.

Hunt's classifications depend largely on the degree of severity and the multiplicity of the ganglionic involvement. He gives as divisions (*a*) herpes oticus; (*b*) herpes oticus with facial palsy; (*c*) herpes oticus with facial palsy and hypoacusis, and (*d*) herpes oticus with facial palsy and Ménière's complex. While he adds sub-groups of (1) herpes facialis with facial palsy and auditory symptoms; (2) herpes occipito-collaris with facial palsy and acoustic symptoms, and (3) herpes of the cephalic extremities with auditory complications.

Using these classifications, herpes oticus is therefore the simplest form of the disease depending on this type of infection of the geniculate ganglion. In it only the external ear and meatus are swollen and inflamed and the deafness of this type is to be distinguished from that due to involvement of the auditory. The next degree of involvement produces facial palsy while its extension brings on the diminished hearing from labyrinthine involvement. Group *d* may be considered as the one showing all the characteristics complete of severe geniculate infection, inflammation, and followed possibly by pressure destruction. In it the inflammatory products may invade the surrounding tissues and extend along the nerve sheath towards the terminus of the nerve. Hunt gives it as his opinion that the ganglions of the auditory nerve itself may at times be pri-

marily involved in the specific inflammation of this disease, but no matter how simple or how extensive the disease manifestations, the symptoms are all dependent on the herpetic infection of the sensory ganglions. Thus, in the group including facial herpes, the eruption is on an area represented by the involvement of the Gasserian ganglion, while the occipito-collaris division has an eruption corresponding on the occiput, side of the neck and the inner posterior surface of the auricle, mastoid, and angle of the jaw, representing the second, third, and fourth cervical ganglions.

Referring to the possibility of a primary involvement of the auditory ganglions, attention may be called to the fact that "cells of the ganglion of Scarpa and the ganglion of Corti take their origin from the neural ridge from which other ganglions liable to zoster are derived. Clinical confirmation of this is found in the occurrence of acoustic symptoms in herpes zoster of the forms with an associated facial palsy. In cases of this kind, if the acoustic nerve were involved by reason of its proximity to the geniculate ganglion, the facial nerve could hardly escape a simultaneous involvement."

Bearing on the matter of the multiple ganglionic involvement, Hunt reports a case of herpes zoster of the third division of the fifth, accompanying which were pains in the external auditory meatus, tinnitus and diminished hearing. McKenzie of London reports a case with aural and facial symptoms associated with laryngeal paralysis. This association may be reasonably expected from what we know of the disease. In this case the first symptoms, noted three weeks previous to his examination, had been slight headache and pricking sensations about the right ear. When examined there had been severe pain in the right ear, swelling of the canal and facial paralysis plus a herpetic eruption on the external ear and the external auditory canal. The drum membrane was normal, as was the region of the mastoid. There was a small group of vesicles on the right malar eminence. In the buccal cavity far back on the right side of the tongue was the erosion of a ruptured vesicle. The whole right side of the pharynx was congested and ruptured vesicles were present on the posterior part of the hard palate and soft palate, but all limited to the right side. Fresh unruptured vesicles were on the posterior right epiglottic surface and the upper edge of the aryepiglottic fold. The right vocal cord was immobilized from right recurrent laryngeal paralysis. Later in this case symptoms dependent on involvement of the auditory nerve arose.

DURATION AND COURSE

The prodromata are estimated to cover a period of from four to seven days before the disease begins to manifest itself in a way that its nature can be recognized. Four to five days usually elapse between the first preherpetic pain and the vesicular eruption. There seems to be no doubt but what the disease may run a very chronic course and the vesiculation persist for a considerable period of time, running into weeks, though the usual evolution will show the subsidence of the eruption in from five to eight days. The stained scar following this will persist for a long period. It is in the symptomatic type of the disease, however, that chronicity is apt to occur and this is not the disease under consideration. It should be remembered that essential herpes is to be considered as an acute specific infection and runs its definite course. The hyperesthesia, which is to be considered a definite part of the disease, can be expected to persist for weeks and in some cases for months after the eruption and its subsequent staining have disappeared.

DIAGNOSIS

The dermatologist will state that the eruption of herpes is pathognomonic and that the disease should be easily diagnosed from the character of the eruption. It should be remembered, however, that these cases do not primarily, or always, reach the dermatologist. The otologist may not always be familiar with the character of the herpetic eruption so that in herpes zoster oticus one cannot be surprised that the herpetic nature of the disease is not quickly recognized. It would seem that a careful clinician should be able to quickly distinguish the herpetic dermatitis and eruption from erysipelas, but it is not surprising that the disease has been diagnosed as mastoiditis; and, simulating mastoid abscess, has been operated on; that sinus thrombosis has been suspected; and, where facial paralysis exists, that labyrinthine suppuration has been suspected. It would seem, however, that if the otologist is familiar, by reading, with the symptom complex of this disease, he would usually readily recognize it. The fully developed clinical picture would be the most easily diagnosed. It is the cases in which the geniculate infection is less marked so that the vesicular eruption is more limited and the associated symptoms of pain, burning and malaise are less marked, that the greater difficulty of diagnosis arises. In these minor degrees of geniculate inflammation, the symptoms

may be limited to a severe otalgia, with or without slight impairment of hearing, and dizziness, making a case still more difficult of diagnosis.

PROGNOSIS

The disease is not to be considered a fatal one, as recovery usually takes place in the course of a few weeks or months. It should be remembered, however, that pain may continue for a long period of time after the disappearance of the eruption. The facial paralysis slowly disappears, though in a few cases it may remain as a permanent lesion. The vesicular eruption gradually disappears, leaving a scaly yellowish brown scar which persists for a long time. Ulceration is not frequent but occasionally occurs, and gangrene associated with it may cause considerable scarring. The usual vesicular cycle occupies from four to eight days. The labyrinthine symptoms are apt to disappear in good part before the facial palsy has subsided, indicating that the auditory involvement is usually secondary to the geniculate infection and may be expected to disappear before the originally infected area becomes normal. Despite a definite statement of the attending physicians, however, the patient and family are apt to feel that the illness is more serious than represented by the attending physicians. The distressing picture of these cases, in which there is full development of the clinical picture, is apt to terrify the family. In my own case, despite a favorable prognosis by all the physicians, the family feared a fatal result.

FREQUENCY OF THE DISEASE

I have personally seen but one fully developed case of this disease in a practice extending over a period of more than twenty years. I believe I have met with minor phases which have not been recognized; that patients who have come to me in consultation, giving a history of pain about the external ear without facial palsy were cases of the lowest grade of geniculate involvement and that other cases in which facial palsy had been present or persisted after the herpetic inflammation had subsided were imperfectly diagnosed as Bell's palsy, the geniculate origin of the disease not being recognized. More than fifteen years ago I operated on a mastoid in a young man who came to me with a history of suffering from a severe pain in the region of the ear and mastoid, but without discharge. He stated that he had been confined to his bed under the care of his physician during this illness and during its course facial paralysis appeared,

which was still present at the time I saw him. There was no evidence of tympanic involvement. There was no diagnostic mastoid tenderness, only a facial palsy and a very slight vertigo. Under the impression that there had been a possible mastoid infection, operation was advised and performed. The cells under the external cortex were found practically normal, but the bone was removed down to the facial canal and in this region an extensive area of hematoma, apparently noninfected, was found in the cells of the bone adjacent to the canal, which was exposed over a fair portion of its course. This patient made a quick recovery from the operative procedure and a short time thereafter the facial paralysis and vertigo disappeared.

From my study of this disease I am now convinced that these symptoms would have disappeared without the operation. Two neurologists of extensive practice in my community have neither seen a typical case of the disease. The same is true of an internist and a dermatologist of extensive practice for a period of several years.

LITERATURE

Investigating the frequency of reports, we find that many have appeared since the reports of Körner in 1904, and Gradenigo in 1907. J. C. Beck of Chicago presented a case to the Chicago Laryngological and Otological Society, April 21, 1914. This patient had been seen several years previously for a peritonsillar abscess. In this illness he had pain in the region of the forehead and about the ear, which kept him awake for several hours of the night but stopped always exactly at 4 a. m. Later there was slight tonsillitis and again the pain stopped at the same hour in the morning. When first seen there was a small red spot in the floor of the external auditory canal which was thought to be due to trauma. A few hours later the patient developed typical facial paralysis and the next morning the herpetic eruption of this disease had appeared. Beck states that this patient also had auditory nerve symptoms, and slight dizziness which persisted for some time.

McKenzie reports a case in the *Journal of L. R. O.*, London, 1915. His case associated the symptoms of aural and facial herpes with laryngeal paralysis and has been spoken of elsewhere in this paper. A. R. Tweedie in the same journal, 1915, reports a case seen in 1911 (note that this report is published after the accumulation of literature on the disease), about October 1; a history of shingles involving the back of the head,

neck, auricle, and also of the left face accompanied by deafness, giddiness, and later (?) by paralysis of the left facial nerve. The nose and throat were normal. He found the herpetic eruption had disappeared. The membrana tympani was opaque with a rough glistening surface. The recovery was slow. He was much improved by the following March and by June had entirely recovered. Note the slow recovery. Another case was that of a physician's wife in which the left ear and its anterior area and the lower apical portion of the mastoid was tender and swollen. Swelling of the canal obscured a view of the deeper portions. Patient had tingling over the back and side of her head with much tinnitus and moderate giddiness. These symptoms gradually disappeared, but in something like a month similar symptoms appeared on the other side. He records this as a case of herpes zoster oticus.

Norman Sharpe reports a case to the New York Neurological Society, June 1, 1915. The patient was of Italian birth, a carpenter, aged 42. Previous history was negative. Illness began with the onset of severe pain in the right ear, headaches, dizziness and a tendency to stagger, and diplopia (origin not stated). A week later there were small pimples and facial paralysis on the right side. Examination showed loss of sense of taste on the right half of the tongue; red spots on the right side of the mouth and right pillars of the fauces. He came to the neurologic clinic for one month on account of the facial paralysis. There was a gradual diminution of the labyrinthine symptoms but slight hypalgesia around the right concha and complete loss of hearing on that side. Two months after the onset taste had partially returned, facial paralysis was still evident, the nystagmus had disappeared.

Dabney, whose monograph on this disease published in the *New York Medical Journal*, February, 1914, is one of the best, reports a typical case. The investigations of J. Ramsay Hunt include a study, however, of a larger number of cases than that of any other observer, and reference to his articles is advised for any one who wishes to make a research of the literature of this disease. He collected the records of sixty cases and reported four of his own. In nineteen of this total there were symptoms of varying involvement of the auditory nerve.

No case of herpes auris or oticus, with facial palsy and auditory symptoms was reported in American literature previous to Hunt's monograph.

TREATMENT

In view of the uncertainty of the exciting cause of the disease the treatment is largely symptomatic. Nothing that I have found in literature suggests anything like a specific or abortive treatment for the disease. It is handled on symptomatic lines, the particular treatment being suggested by the ganglions involved, and the completeness in evolution of symptoms corresponding with the severity of the infection. Naturally intestinal cleansing and a light diet are called for in the early stages of the disease, with narcotics sufficient to give the relief that can be obtained in this way. Later zinc phosphid, arsenic, galvanism, and other tonics are indicated to be used. In view of the progress made in serum therapy it would seem worth while in these cases to investigate the bacteriologic flora of the tonsils or any other focal infections in the upper respiratory tract or about the teeth, and use a treatment based on the findings of this investigation.

I wish to present the report of a typical case occurring in my own practice, which shows a very beautiful symptom complex of this disease so that an exposition of its course is a classic résumé of geniculate involvement with secondary implication of the vestibular and cochlear branches of the auditory.

REPORT OF CASE

Sept. 25, 1916, I was called in consultation to see Mr. S. A. N., aged 70. Ordinarily, he was a man of marked physical activity and energy and seemed much younger. He occasionally showed signs of intestinal toxemia. He had been seen by his physician, Dr. B. E. Lindsey, four days previously for the first time. At that time he was complaining of burning and pain in the region of the left ear, with slight dizziness. He had had a moderate postnasal inflammation a short time previously, and had a moderate bronchitis at this time. His temperature was normal, blood pressure was low, 95, Wassermann test, negative; urinalysis negative; but the patient felt miserable from his illness. The day on which I saw him, four days after the first visit of his family physician, there was an increase in the burning and distress about the ear and an inflammatory swelling showed, not only in the canal, but directly below the external ear. Hot moist compresses had been used to relieve this. I found an intense dermatitis in the canal and a few vesicles. There was a beginning facial paralysis affecting the muscles supplied by the facial except the orbicularis. Intestinal cleansing treatment, which had been begun, was continued and an ichthyol ointment used locally. I saw the patient again October 6 when the entire symptom complex of Ménière's disease had appeared in addition to com-

plete facial palsy and more marked signs of local inflammation about the canal. The palate did not show involvement of its facial anastomoses. At neither visit, however, was there any involvement of the deep portion of the canal or of the drum membrane, which appeared practically normal. In the height of the labyrinthine involvement there was total deafness in the ear, vomiting was spontaneous, and later was induced by slight movement of the head or jarring of the bed when any one walked across the floor. These labyrinthine reactions appeared nearly three weeks after the first indication of illness and persisted for three weeks when they gradually subsided. There was entire absence of acid in the stomach contents during the height of the reaction. By November 1 the improvement became rapid, the labyrinthine symptoms first yielding and later the facial paralysis clearing. Jan. 2, 1917, the patient was seen at my office, at which time the facial paralysis had disappeared, though there was still vague discomfort and abnormal sensitiveness in the region about the ear. A hearing test showed complete restoration of function. Tuning fork 128 v.s., heard in both ears equally; bone conduction, 45; air conduction, 75; 2,048 v.s., both ears, air conduction, 10; watch, both ears, c/50.

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DISCUSSION

DR. J. A. LICHTY, Pittsburgh: I feel myself highly honored to be called on to discuss so scientific a paper and so rare a disease. I find myself very much in the same happy position in which the lawyer found himself who belonged to a shooting club. He was not much of a solicitor, nor much of a shot, but the lawyers all thought he was a good shot and the men in the shooting club thought he was a good lawyer. I have listened to the discussion this afternoon and it struck me as very interesting. I cannot say I have understood it all, but I want to express my indebtedness to men of this character as I see you have in your Academy for the kind of work you are doing and for the assistance you are to the internist in ferreting out these difficult cases which come to one with their own diagnosis of neuralgia. I am afraid I cannot add much to the discussion, and will have to confine my remarks more generally to the subject of herpes zoster. When Dr. Brown asked me to discuss this paper I thought I had had a large experience in herpes zoster, but in looking over my records I found I had seen about seventy-five cases in my medical experience, and this was taken from about

16,000 patients. I have no doubt that some of the 16,000 patients may later have had herpes zoster of which I did not hear. I wish to say that seventy-five in 16,000, or one-third of 1 per cent, would represent the incidence of herpes zoster. I have not had the fortune of seeing just such a case as was described by Dr. Brown. The interesting part in these cases, of course, is the etiology. The etiology of herpes zoster as you go down through the development of our knowledge of the disease which has been very rapid in the last decade or so is rather a pitiable story. We began first by considering these cases as neurasthenics and they of course developed herpes zoster. I remember the first two cases I saw. One was in a woman who had trouble in clearing up an estate and at the end of the year she developed an attack of herpes zoster. Another was a bank president who I found out later was losing his fortune, and during that time developed an attack of herpes zoster. Now later as our knowledge developed we looked on herpes zoster as a manifestation of "rheumatism." You know how broad a term rheumatism was considered in our early days. We hardly speak of it now. We first divided our cases into rheumatism of which we knew nothing and then acute rheumatic fever of which we thought we knew a great deal, but now with focal infection we hesitate to speak of these patients as suffering from acute rheumatic fever. So in herpes zoster we have come through that same line of development until now we look on herpes zoster, as the writer suggested, as being due to infection which is probably our last step. Rosenow has given us a great deal of information. He demonstrated on dogs and other animals that the posterior root ganglions are infected. It is made so easy and plain by him that you really hesitate to take the whole thing as he presents it to you. This has not been worked out in man. In this case presented by Dr. Brown, the inflammatory condition found in the mastoid may have been an infection which produced the symptoms and which probably would have developed into a herpes zoster if he had not operated on the patient.

With reference to focal infection we have gone a long way and yet I do not believe we know all about it. I am very much impressed with Adami's work in this line, in which he speaks of the fact that we have in our tissues certain microorganisms which at the time are not virulent, but which under favorable circumstances become virulent and produce the lesions and give the symptoms which we find. In our own city here at the University of Pittsburgh Dr. Holman has worked this out beautifully in the lower animals in which he found the microorganisms cultured from most any tissue, so I do not believe you can always conclude that when you have a focal infection that the lesion is due to that. So much as to the etiology. While I believe it is an infection I do not know that we are always sure that the focus which we find is the specific cause of the lesion. The diagnosis is a very interesting point. Dr. Brown brought out one point which is important and that is the pain which comes before the skin lesion appears. I remember a patient who thought she had heart trouble and in tracing the tenderness along the ribs we traced this back to the spinal column and found considerable tenderness to the left of the spinal column. She later developed a herpes and her heart was normal. Another point is the chronicity. Even though the lesion has healed the pain may last a year or longer. I saw a patient in whom it lasted two years. As to treatment I have very little to say. Nothing can be done, but locally I have applied colloidion to the lesion morning and evening which relieves the pain.

DR. JOSEPH C. BECK, Chicago: The case referred to by the essayist which I had of herpes zoster oticus was this: A man about 200 pounds

in weight, perfectly healthy, gave a history of repeated peritonsillar abscesses which always ruptured spontaneously. At this particular time he developed a peritonsillar abscess which did not rupture. He came to me saying that he had a sore head, particularly in the ear, a burning sensation. I explained to him that his pain came from the peritonsillar abscess. About three-quarters of an hour afterward he telephoned me to come over to his place of business stating that everything he ate fell out of his mouth. One glance showed that he had a facial paralysis. I never saw a case come on so quickly except perhaps after operation on the mastoid. At this time he had no herpes, but on closer examination of the pinna and external canal, I noticed a red areola around a black spot. The next day when I came to his house he had the beginning of three little vesicles on the auricle. He developed a marked tinnitus, vertigo, spontaneous nystagmus and the next day a complete deafness on the affected side. The patient has never completely recovered from his facial paralysis. He has a paralysis of the orbicularis palpebrarum which is constantly twitching. He has never had his tonsils removed and he has had no recurrence. I told him that one attack is said to confer immunity from the disease and so he is willing to have his recurrent peritonsillar abscesses.

DR. J. H. FOSTER, Houston, Texas: I think the condition described by our essayist is more frequent in occurrence than is generally believed. I became interested in this subject some years ago when Dr. Hunt published his monograph on involvement of the otic ganglions.

I have seen several typical cases and a number of mild degree. One patient in particular had attacks of varying degree over a period of several years which had on occasion been diagnosed as middle ear abscess. The pain in these attacks was excruciating. After the removal of her tonsils she has been free from recurrences.

Three weeks ago I saw a typical case coming on with pain, then developing tinnitus, dizziness, deafness and facial paralysis. The family physician was loath to believe that there was not an infection of the ear on account of the pain and extreme tenderness of the canal of the ear which was the seat of an eruption of herpetiform nature.

DR. W. LIKELY SIMPSON, Memphis, Tenn.: I will mention a case I had a year and a half ago. A little girl aged 6 or 7 years, gave a history of pain the week before I saw her. There was, also, a slight watery discharge and some redness of canal. When I saw the case, there was a normal drum. Several red points, which, no doubt, had been blisters were located on the posterior wall of the canal, and, also, on the auricle. There was no hearing (noise apparatus in left ear), and no response to the caloric and turning tests. The nystagmus suggested only a functioning left ear. The hearing, caloric, etc., in left ear were normal. There was complete paralysis of the right side of the face. The child was rather small, 6 years old, and some of the tests could not be made, but, so far as I could make out, the case was typical of herpes zoster oticus.

The hearing came back almost to normal. The vestibular portion of the labyrinth, also, came back to normal.

THE USE OF KINESTHESIA IN TREATMENT OF SPEECH DISORDER

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BOSTON

INTRODUCTION

Thirty-five thousand cases of speech disorder in France alone last year is a fact that will move you to take some interest in this subject.

All specialists correct and perfect the functions in their several fields. As a function in the field of laryngology, speech correction belongs to you. Twenty years ago the pathologist took little if any interest in the nervous system. Laryngologists of today look askance on speech disorders in much the same way.

As a speech disorder specialist, I have more to do with the functions of the throat than you do. After twenty years of study on this subject, I come before you to try to unify these two fields. I hope therefore you may hear papers on this subject every year, as it is a function of your specialty which you have grossly neglected.

My first attempt at this unification is this paper of today.

ANATOMIC REVIEW

The kinesthesia of speech can best be explained by reference to brain anatomy. Recall, for a moment, the left hemisphere of the brain in the region of the fissure of Rolando. Anterior and posterior to this fissure are two long convolutions that run up the entire side of the brain surface. The anterior convolution is called the *ascending frontal*, and the one behind the fissure is called the *ascending parietal*. The anterior convolution subserves the function of motion and is divided into thirds; the upper third governs the motions of the leg, the middle third those of the arm, and the lower third those of the face. The posterior convolution is likewise divided into thirds; the upper third receives the sensations of touch from the leg, the middle third those from the arm and the lower third those from the face.

Suppose, now, that I say to you, "Close your eyes and place on the table the smaller of the two coins which I hand you." If you perform this correctly, you have sensed the coin in the central third of the ascending parietal convolution, and have interpreted this sensation in the cortex just posterior to the central third, and have made a motion with your arm in placing the coin on the table which is governed by the central third of the ascending frontal convolution. In other words, you have gone through the following functions: you have received a tactual sensation; interpreted this in a tactual interpretive area; and, made a motion guided or dominated by that tactual sensation alone. You have acted under the dominance of a tactual sensation guided by a tactual interpretation. Your functions have been kinesthetic.

It is clear, then, that when kinesthesia is used in treatment of speech disorders, the functions of only two or three neighboring convolutions are used. Speech is enervated from the lower third of the ascending frontal convolution and the tactual sensations which guide this motor output of speech come through the lower third of the ascending parietal convolution. The whole process is carried on, in both its sensory and its motor aspects, by these convolutions alone.

Now, the discoverer of diaschisis tells us that when a brain injury shows certain local symptoms there are also, correlated therewith, other symptoms which come from other areas in the brain connected by fiber tracts. If we turn this about and apply it to the field of speech correction, we get the following formula: In working on the brain between only two convolutions one has less chance of diaschitic development than in working on areas whose connecting fibers go to more distant and various convolutions. Stating this in anatomic terms, there is more chance of diaschitic development in working from Wernicke's to Broca's convolution or from the cuneus out through Broca than in working from one convolution to the next nearest. And yet this latter is all that is accomplished in working from the lower thirds of the ascending parietal and the ascending frontal convolutions.

Let us carry these ideas over into the field of speech disorder. When we ask the speech patient or pupil to concentrate attention on the positions of the speech organs during the enunciation of given sounds and train him to follow the guidance of these tactual sensations alone, we are working primarily with kinesthesia.

While I do not wish to deny the usefulness of this method, I may point out that it is necessarily partial and incomplete in its results. The exclusive use of it will not serve our purpose. No one can afford to neglect it, but it must be used in combination with other and more important methods. And this is for the reason just given, that it calls into play so limited a part of the brain cortex.

SPEECH CORRECTION

In spite of the present popularity of the kinesthetic method and in spite of the large number of practitioners who depend primarily on it, one may well ask whether, from the standpoints of anatomy and advanced psychology, we may not discover other and more fruitful methods of speech correction. May we not in our speech training call other areas of the brain into action? Are there not other mental contents that may and should dominate speech production? Have not the practitioners who deal exclusively with the kinesthetic method left out a large part of the necessary mental background? I think these questions may all be answered by an emphatic affirmative.

We all know, for example, that the acquisition of language is largely a matter of the ear. Even grammar, a minor detail of language, is so learned. In the matter of tone quality or *timbre*, the mere listening to great vocalists establishes in the minds of the hearers a lofty vocal ideal which affects their own voices. These considerations would imply that aural intake and registrations form an indispensable background in all vocal utterance. Over and above the kinesthetic element in speech, therefore, there must be an acoustic mental content which permanently dominates utterance.

But this is not all. It has been shown in recent investigations that in the enunciation of a word there is always present in the mind of the speaker a concomitant mental picture or visual image. Some sort of visual content pervades the mental horizon during all normal speech. But, this extremely important fact is simply ignored in the pure kinesthetic method. It is inexcusable for the speech expert to neglect the training of these visual processes. Because we find that these visual processes constantly accompany and to a large extent control all normal speech, we are justified in saying that any method of treating abnormal speech which ignores them is markedly incomplete.

Nearly the same thing may be said of the utilization of the collaborative processes. I mean by "collaboration" any use that is

made by the mind of the sensorial intake, such as reasoning, reflection, meditation, emotional reaction, and the like. Some collaborative process, whether simple or complex, is usually present and dominant during speech. Since these processes or mental attitudes are present during normal speech, it is obvious that they should be deliberately instilled into the treatment of abnormal speech, in order that the speech background may be made as nearly normal as possible. But the kinesthetic worker ignores this. He is content to teach the production of sounds through the sense of touch and muscular effort, through the feeling of mouth positions, to establish in his patients the habit of sensing these positions tactually and then reproducing them by kinesthetic memory. He never attempts to build up over this kinesthetic memory a collaborative content; he never attempts to govern kinesthesia by the imagination. Accordingly, he never can enable his patients and pupils to exchange the humdrum utterance of the kinesthetic memory for the refined and polished utterance which is governed by complex emotion and thought.

Although it is true that normal speech is to a certain extent kinesthetic, acoustic, visual, and collaborative processes in all their numerous forms and combinations play a still greater part in the guidance and control of speech. Indeed, it may be said that any speech correction method which relies solely on kinesthesia is employing about one-fourth, to speak generously, of the normal mental background of normal speech. This is indeed a very generous estimate, for it leaves out of account the argument from diaschisis which I have referred to. Diaschisis sets up a rapid cross-fertilization among the various powers and functions of the mind which makes the employment of four sets of faculties not merely four times as good as the employment of one but almost infinitely better. Diaschisis provides the most cogent argument in favor of building up the background of speech on the basis of the entire cortex by employing all the psychologic functions that can be put behind speech.

SUMMARY AND RECAPITULATION

Let me say again that kinesthesia of speech may be defined as tactual sensation governing vocal output. It is anatomically located in the lower third of the ascending parietal convolution and takes its exit from the lower third of the ascending frontal convolution. Some speech experts consider it sufficient to develop these areas alone, but when we study the background of normal speech we find that it has acoustic, visual and collabora-

tive, as well as kinesthetic, contents. Since these are found in all normal speech, it is necessary to keep them in mind in all speech training and to develop them in those abnormal cases where they are found lacking. In anatomic terms, it is indeed necessary to bring to normal development the functions of the lower thirds of the ascending parietal and frontal convolutions, but it is equally necessary to develop the acoustic functions of Wernicke's area, the visual functions of the cuneus, and the collaborative functions of the other brain parts before anything like normal speech can be attained.

A NEW EAR TEST FOR MALINGERING

FREDERICK F. TEAL, M.D.

LINCOLN, NEB.

In examining men who were included in the draft, I have encountered some difficulty in uncovering the dishonesty of "slackers" who claim to be deaf in one ear; the Weber and Rinne tests are a great help, but even with these it is at times difficult to be certain that a recruit is or is not deaf in one ear.

For these instances I have used the following reliable test which, in the absence of any record of it as far as I am able to determine, I believe is original with me. The test is of service in unilateral catarrhal deafness only, but as this comprises the greater number of ear malingerers it is exceedingly useful.

The person is blindfolded and in a friendly; helpful manner told that if he is really deaf there is no disposition on our part to overlook it. But he is also warned that if he tries to show dishonesty in the test, he is sure to be "tripped up" and his veracity doubted.

Air conduction is tested and of course is negative. The Weber test is then used and usually (though reluctantly) he hears the fork in the deaf ear. Bone conduction over the mastoid is next tested and again he admits hearing the fork. He is then commended for his answers and assured that he has answered as he should.

The last and real test is now used. After saying you want to try the last test (bone conduction) once more, a non-vibrating fork (or a lead pencil—flat end) is placed over the mastoid to make him think he is being tested in the same manner, but at the same time a vibrating fork is brought up close to the auricle with the other hand, to test the air conduction. If he is simulating deafness he will of course answer that he hears the fork (under the impression that he hears the sound through the bone) and the fact of a normal path of air conduction is established. If he is really deaf, he will of course not hear the vibrating fork.

TRANSACTIONS
OF THE
TWENTY-THIRD ANNUAL MEETING

OF THE

American
Academy of Ophthalmology
and Oto-Laryngology

OPHTHALMOLOGICAL DIVISION

HELD AT
DENVER, COLO.,
AUGUST 5 AND 6, 1918

A CLINICAL CONFERENCE

NEW METHODS FOR TREATING CHRONIC DACRYOCYSTITIS

DR. MELVILLE BLACK, Denver: When I began the study of ophthalmology I soon discovered that the treatment of dacryocystitis was far from satisfactory and I am sorry to say that it is still unsatisfactory.

Those who are in favor of syringing through the unslit canaliculus over a considerable period of time have insisted that they get results which are worth while. All I can say is that I cannot get them. In private practice, one hesitates to keep patients coming until large bills have accrued and then have to strike them off the books or cause dissatisfaction and hard feelings if payment is insisted on. In my clinic I have tried it thoroughly, but it has not given results.

In suppurative cases in which the patients are exposed to abrasions of the cornea from foreign bodies and like injuries and who cannot afford to come for any form of treatment which would take them away from their work several times a week, it has been my practice to destroy the lacrimal sac. At first I performed the operation of extirpation, but I gave it up for Gifford's operation, which I have found equally as satisfactory and much less difficult to perform.

The operation of resection of the lacrimonasal wall has always appealed to me, and I have referred several cases to rhinologists who were doing it. Some have been successful and others failures. It may be that we did not keep up the probing long enough after the operation. We have been doing this of late and these cases have done better. It is evident that only those whose time will permit can enjoy the benefits of this operation. Even though the window closes, I have noticed that supuration did not always return. I devised knives for removing the lacrimonasal wall after cutting down into the sac through the skin. This method was not satisfactory, probably because the two raw surfaces on each side of the sac caused adhesions which led to closure.

What we want is an operation that will give quick and permanent results. Dr. Thompson of Texas proposes curettage of the lacrimal duct. The procedure does not sound rational. It does not sound like a good surgical principle to curet the mucous

membrane of the lacrimal duct. It would seem as though adhesions must form between the lacerated walls of the duct and a complete closure of the passage result. I reasoned, however, that if it did, the patient would not be rendered any worse and that a window resection or destruction of the sac could be performed just as well or better if the lacrimal duct had been obliterated. I therefore decided to try it. I have only operated on two cases, and they have not stood the test of time, but the results so far are satisfactory, and I shall continue to do it until I have satisfied myself as to whether it is an operation of merit or not.

DR. JOHN GREEN, JR., St. Louis: I want especially to speak of Dr. Thompson's operation which he described in a paper read before the section on Ophthalmology of the American Medical Association in June. His argument is that dacryocystitis is an end-result of an occlusion of the lumen of the nasal duct, which is brought about by the deposition on the walls of the canal of solid detritus carried down by the tears. He points out that the duct is lined with nonciliated epithelium in the form of folds, which readily catch and hold this foreign matter. Erosion occurs, followed by the deposition of salts derived from the tears and serum, leading finally to the creation of a mass which completely plugs the canal. It is rational, then, to scrape off this mass from the walls of the duct by a process of curettage rather than to push it through with probes. Whether this theory is correct or not, it is at least plausible and worthy of careful consideration.

Dr. Thompson first slits the canaliculus freely and injects cocain into the sac. He then injects hypodermically a weak cocain solution. A small sized Buck's ear curet is passed to the entrance of the bony duct and then by a rotary motion accompanied by gentle pressure it is made to find its way down to the nostril. The process is repeated with the middle and large sizes. An applicator dipped in 25 per cent. tincture of iodine is then applied to the sac and duct.

Since the pre-session pamphlet came out I have operated on eleven cases by this method, and Dr. Jacobs, my colleague, has operated on three.

I agree with Dr. Black that it is better to do a preliminary probing before attempting to introduce the curet, thus minimizing the chance of creating a false passage. In my first cases I rapidly dilated up to 11 or 12 Theobald, then injected 10 per cent. cocain-adrenalin. I now use smaller probes, my aim being simply to create a passage for the adrenalin-cocain solution,

which is repeated three times before the curets are used. Using the stronger cocain solution, I have found it unnecessary to inject cocain under the skin. The procedure is quite bearable, if not entirely painless. For the application I have used "iocamfen," which is less corrosive than tincture of iodine. In the main, results have been satisfactory, though, of course, too short a time has elapsed to speak with absolute confidence of lasting cures. I should like to cite three cases which are fairly typical of what may be expected in cases of chronic dacryocystitis.

CASE 1.—Mrs. W. B. has had a chronic dacryocystitis a long time. Pus can be expressed. June 25, Thompson's operation was performed, and the case was progressing smoothly August 1. No pus can now be expressed and there is a free passage into the nose.

CASE 2.—Mrs. F. E., aged 60, treated in the clinics with probes for months without improvement. Thompson operation was performed June 5. July 1, passage free to nose, no pus can be expressed.

CASE 3.—A. C. had chronic dacryocystitis. Thompson's operation was performed June 1. Dilated rapidly to 13 Theobald. The patient now is free from pus in the sac and there is no stillicidium.

Not all of my patients have done as well as these three, but all have shown definite improvement. I shall repeat the operation as advised by Dr. Thompson in those cases that have not yielded to the single curettage. My experience with the operation is distinctly encouraging and I propose to continue to perform it.

DR. F. F. TEAL, Lincoln, Neb.: I had the pleasure of hearing Dr. Thompson's paper, and have carried this treatment out with the experience of Drs. Black and Green. I have had four cases similar. All I had probed for months. The first was the wife of a physician, and I stated to him that I believed that we had something good and new, and I would like to try it on his wife. I followed the procedure as outlined by Dr. Thompson, and on the second visit I passed a Number 12 Theobald probe and have never had to do anything since.

In the second case I did not have quite such good success. It still discharges pus. I used a larger probe and eventually got in with a Buck's curet and discovered some rough points around the ring not removed by the first operation. These I cleaned up as well as I could at the second operation, and so far have had perfect results for several months. I attribute my failure the first time to the fact that I did not curet thoroughly. It is surprising how large a probe you can get in after this curetting process. The hardest thing is to apply the tincture of

iodin. I tried Dr. Thompson's method of winding a probe with a thread, but on account of the blood and serum it did not seem to me to get thoroughly in contact. I believe Dr. Thompson has shown us something of merit. I have four cases, two of which are classed as cured; the two others have done well and I believe will be cured, and that is more cases than I have cured since I have been in ophthalmology, not including, of course, operations on the sac.

DR. WILLIAM H. CRISP, Denver: I have not had a long series of cases, but in several cases in which the trouble with the tear sac had gone on for several years, and especially in some in which a slitting of the canaliculus had been done previously, I have obtained very good results from a thorough slitting of the canaliculus back to the internal canthus. I used a very narrow canaliculus knife, holding the lid on the stretch by means of a pair of chalazion forceps, for want of any other convenient instrument. It seemed to me that in some of these cases we had the result of an anatomic arrangement in which drainage was easily interfered with, so that if we gave better drainage the infection of the sac would clear up without further operative procedures.

DR. HAROLD GIFFORD, Omaha: Just because I have a method of destroying the sac, I do not want to give the impression that I use it in all cases of dacryocystitis that are examined by me. On the contrary, I am very conservative and try to restore the duct in the great majority of cases. The destruction of the sac as a treatment of dacryocystitis is very old. Years ago I used to do it with the Paquelin cautery with first rate results, but as there was always some fear in the back of my mind that I might cause necrosis of the bone, I substituted trichloracetic for the cautery. The former has the advantage that its application immediately produces a protective eschar so that it is almost impossible to go too deep with it.

With regard to Dr. Thompson's method *a priori*, it seems to me entirely wrong to curet the canal. I should expect such treatment to cause the canal to close up instead of to remain open, but the evidence in its favor seems to be increasing and I feel inclined to try it after what has been said here today.

I have been asked whether I ever use the trichloracetic method in acute cases of lacrimal abscesses. I have not tried this, and I believe it is better to open the abscess and keep it drained until the inflammation subsides under the use of hot applications before proceeding with the destruction of the sac.

With regard to argyrol and other silver preparations, I regard them as poor agents to use in the lacrimal passages. I have seen two permanent black eyes as the result of using argyrol after attempts had been made to probe the duct.

DR. D. T. VAIL, Cincinnati: The essayist has covered the ground so completely that there is very little more to be said on the subject, and yet one of the most striking forms of acute dacryocystitis, namely, the phlegmonous type, deserves some further attention.

Whether due to recrudescence of an old suppurative process or a fresh infection, phlegmonous dacryocystitis takes on many of the symptoms of erysipelatous abscess. There is very pronounced swelling, considerable fever and marked pain; in fact, all the symptoms of a localized acute septic infection. The surgical measure that I have adopted for this pronounced form of dacryocystitis is, in principle, identical with that which many surgeons employ when dealing with carbuncle of the neck or so-called "anthrax." The necrotic area is surrounded by a deep incision and the mass is extirpated during or just after the height of the storm. The patient is put under a general anesthetic and the tear sac or abscess cavity is removed by rapid and bold surgery, paying no attention to the anatomic arrangement of the layers of tissue and disregarding the hemorrhage which is always profuse until after the mass is excised. A deep curvilinear incision, such as Axenfeld recommends, is carried through all the tissues including the periosteum, to the bone on the side of the nose, the lacrimal groove is found with the periosteotome, the strong tissue-forceps or mouse-tooth forceps are made to grasp the tissues lying in the lacrimal groove which we know to be the lacrimal sac, and with good curved scissors the dissection of the sac and contents is completed by intelligent and bold clipping. The operation is completed inside of twenty minutes, including the application of deep interrupted sutures. I have no patience with the German method, beautifully illustrated in Meller's "Surgery of the Eye," where you are to look for twelve layers of tissue, one after the other. It seems to me like "puttering" and is a waste of valuable time. Extirpation of the lacrimal sac is not an operation similar to the operation for inguinal hernia, for the different layers in the former may be entirely ignored. The recovery after extirpation of the abscess cavity is prompt. The cases I had all recovered without incident or deformity, usually within two weeks' time. I think this is a valuable procedure and deserves a place in the consideration of the treatment of this form of dacryocystitis.

DR. J. C. BECK, Chicago: This is, perhaps, the field of the ophthalmologist, and yet the tear organ is usually the borderline in which the discussion of the newer methods of treating dacryocystitis comes in. I have never done the Thompson operation, but did the West operation in a number of cases, so a confession might be of value. The earlier cases operated by this method were absolute failures so far as results were concerned, and so I operated others by the method advised by the modification, and in the last three cases the results were excellent. The point is in making a large enough opening that it will remain open. This was done by a protected bur, and I would like to add this to the discussion.

DR. LUTHER C. PETER, Philadelphia: Dr. Gifford has pointed the way to classify chronic dacryocystitis into two groups: (1) those which should be treated radically, and (2) a smaller group which should be treated tentatively. If the punctum has been slit open it will never again functionate properly, and as a rule there will be a reinfection. The unpleasant symptom, epiphora, will continue even though a reinfection does not promptly take place. When the case is well marked and of long standing, extirpation of the sac as described by Dr. Vail should be done, and in all cases in which the surgeon finds it necessary to slit up the canaliculus, I believe it to be a better policy to resort to the more radical extirpation. The pathology of these cases begins at the lower end of the sac in the lacrimonasal duct. In a few instances the obstruction may be found in the nasal chamber, as Dr. Beck says. Therefore, many of these cases might be saved more radical surgery if the nose and throat man is called in to explore and relieve the obstruction of the nasal end of the duct. Slitting up of the canaliculus should be avoided in the treatment of these cases.

DR. H. L. BAUM, Denver: Although this is primarily an ophthalmologic subject and I have had no experience in treatment of the condition by other than intranasal means, I have done a number of intranasal operations for lacrimal drainage and wish to add a word in regard to that method. Dr. Beck has said that the results following intranasal operations have not been 100 per cent. perfect, and such has also been my experience, but I think we can claim success in 75 per cent. of our cases, when the operation is properly performed and postoperative treatment faithfully carried out. The main point, beyond removal of sufficient of the internal wall to secure an adequate opening into the nose, is postoperative treatment to prevent the formation

of granulation tissue and subsequent closure of the artificial opening. Frequent applications of silver nitrate solution and regular probing will usually prevent this undesirable termination.

If we can cure these cases by the intranasal method, whether a West operation or a modification of it is used, we will do away with the slitting of the canaliculus, with considerable advantage to our patients and some advantage to ourselves. One of my recent cases occurred in a child of 16 months, referred to me by Dr. Libby. From the history I concluded that the lacrimal infection had occurred secondary to an acute conjunctivitis, and supuration had existed for some months. At the time of examination a fistulous opening was present from which there was a free discharge of pus, with extensive excoriation of the skin of the lower lid. The fistula had followed an attempt to perform the Gifford operation, which had been unsuccessful. My procedure in this case was the intranasal drainage by means of a simplification of the West operation described by me two years ago, in which I use a curet for removal of the lacrimonasal wall and the internal portion of the sac. Any operation was rendered difficult in a child of that age, and I believe the West operation would have been impossible. The immediate result was good, and on the sixth day the fistula had healed. As her home is not in Denver I have not seen her since that time, but a letter from her parents received this week states that she has remained well, with no indication of further trouble excepting an occasional slight epiphora. It has now been nearly five months since the operation.

GRANULOMA OF THE CORNEA

M. FEINGOLD, M.D.

NEW ORLEANS

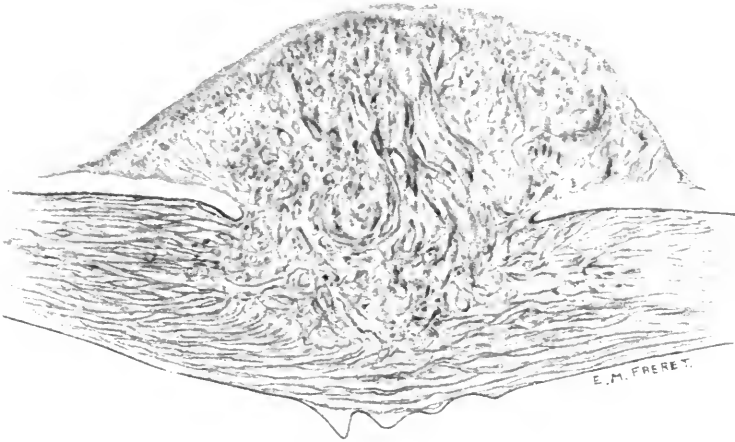
Possibly no structure of the body has been the subject of so much study, clinically and especially experimentally as, owing to its peculiarities, the cornea. But in spite of the many varieties of corneal affections described, and though the process of healing of wounds of all kinds has been studied and recorded minutely, one fails to find, even in the best and most modern textbooks, any mention of granuloma formation of the cornea. For this reason, the following cases seem to deserve recording, and a review of the few cases reported in the literature might not be amiss, especially since some of the case literature is not easily accessible and because the condition is generally placed among the tumors of the cornea instead of among the inflammations and their consequences.

REPORT OF CASES

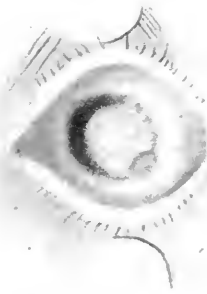
CASE 1.—H. J., colored, laborer, aged 56, applied at the Charity Hospital Eye Clinic, Nov. 9, 1917, stating that, three weeks before, muriatic acid had splashed into his eyes and that the right one was so badly injured by it that enucleation had to be done two weeks later. The conjunctiva of the eyeball appeared reddish white, the whole cornea grayish, and he could notice hand movements. Gradually a grayish-red soft granuloma 3 by 3 by 2 mm. appeared on the conjunctiva of the eyeball at the lower temporal limbus, and a similar flat one 3 by 1.5 mm. at the upper temporal limbus.

Dec. 6, 1917, the lower granuloma was seen riding on the limbus with its greatest portion on the cornea. The smaller granuloma was wholly on the cornea near the upper limbus. The cornea appeared at first glimpse quite clear in the peripheral portion, but this was due only to invasion of superficial and deeper vessels from all sides. Through it, the iris could be guessed at. The central portion was whitish opaque and slightly depressed, with irregular surface. There were good light projection and color perception.

Later the upper granuloma moved 2 mm. from the limbus to the center getting smaller at the same time and appearing more red; the lower granuloma also grew smaller and more flat. The central area of the cornea became more bluish white and smaller, because encroached upon by the increasing ring of vascularization; the thickened grayish-red conjunctiva of the eyeball extended to the cornea forming a false pterygium in



Case 1.—Granulomas of the sclerocornea.



Case 2.—Granuloma of the cornea. Mallory's connective tissue stain; $\times 24$.

several places. This was especially noticeable at the seat of the lower granuloma, which in the meantime had moved away from the limbus, leaving a narrow band of cornea between itself and the limbus. The upper nasal portion of the cornea cleared perceptibly and the patient could see faces.

Three months after the accident, the lower granuloma was smaller, triangular, and apparently pedunculated while the upper granuloma had entirely disappeared. During the further observation even the lower granuloma faded away gradually and the whole cornea cleared sufficiently so that, about May 1, 1918, patient could read 2-inch letters close to the eye. During the whole time the tension was carefully watched and always found to be below normal on palpation. Only once did the tension seem to be increased. Treatment consisted only in inspersions of ethyl-morphin hydrochlorid powder (Figure 1).

CASE 2.—E. B., a farmer, aged 42, called at the Charity Hospital Eye Clinic, Sept. 29, 1915, because of inflammation of his right eye, which had begun when some unslacked lime got into his eye three weeks before. The eye showed moderate ciliary injection; the corneal epithelium was markedly defective, the cornea itself diffusely nonsaturated, bluish, opaque with a deep-seated round 3 mm. gray infiltration slightly below the center. There were 3 mm. of hypopyon.

The hypopyon soon became absorbed but recurred several times. Gradually a small, soft, reddish-gray tumor of granulation tissue appeared near the center of the cornea. Acetylsalicylic acid, ethyl-morphin hydrochlorid, argyrol, atropin, and physostigmin (eserin) were used to meet symptoms and indications. Repeated attacks of severe pain caused by increased intraocular tension finally necessitated enucleation which was done under local anesthesia, March 20, 1916. The eyeball was immediately placed in Zenker's solution, after hardening bisected in the equator and sectioned horizontally by Lee's dry celloidin method.

Histologic examination.—In the conjunctiva only a few plasma cells are seen, but the epithelial cells have a tendency to desquamation. The cornea is slightly pressed in toward the anterior chamber in the central part. This was apparently caused by the lid bulging the softened cornea in, when pressing on the projecting growth. The cornea is about 0.6 mm. thick in the periphery, somewhat thicker in the central portion. Here a mushroom-like growth is lying on the surface and is continuous with the corneal substance by its very short pedicle. The epithelium is missing on the whole corneal surface. At the limbus the epithelium of the conjunctiva projects for a short distance over the cornea, but is not in contact with it, forming a folded band which often folds back on itself toward the limbus.

But for an isolated, cell-like structure here and there, Bowman's membrane is entirely bare of epithelium. It is continuous over the whole surface of the cornea, except for about 0.98 mm.

in the center corresponding to the pedicle of the growth. It is about 16 microns thick and seems to consist of two portions. The superficial one is about 3 microns thick and stains rather deeply with hematoxylin and purplish with Mallory's connective tissue stain. The posterior portion stains more like the corneal substance. The anterior portion splinters off here and there from the underlying portion to some extent. Bowman's membrane, and to a slighter extent the adjoining lamellae of the cornea, contain numerous, small, amorphous and more crystalline, yellowish brown particles of different size (argyrol?). Bowman's membrane is everywhere in normal contact with the underlying parenchyma. In some sections, near the pedicle of the growth, masses staining intensely blue with hematoxylin are seen between Bowman's membrane and the parenchyma and also in the spaces between the next three or four layers adjoining. These masses are fine and linear or more lumpy and often cover the corneal structure entirely (lime?). Vessels pervade the whole cornea, lying mostly in the anterior two thirds. A few smaller vessels are immediately below Bowman's membrane. In the immediate neighborhood of all the vessels are seen cells, each with a long nucleus, staining deeper than the corneal cells, also an occasional eosinophil. The whole cornea is seemingly unaltered except that the fixed cells are evidently more numerous than normal. Higher magnification reveals some of these, at least, as possibly wandering cells, since their nuclei stain deeper blue and since two or three cells are at times seen in the same space. Except for the affected area, the cornea shows no differences in the arrangement and appearance as to color, etc., of the lamellae, in spite of the marked opacity *in vivo*.

The affected area is about 2.8 mm. in the largest diameter and is consequently larger than the defect in Bowman's membrane. It is roughly lenticular in cross section, that is, thickest in the center. It occupies the middle layers of the cornea, leaving the posterior fourth entirely free. Its edges are entirely in the deeper layers with comparatively unaffected corneal substance in front of it. Over the central portion, on the other hand, corresponding to the defect in Bowman's membrane, this area extends even through the anterior layers, and is here continuous with and forms the pedicle of the mushroom-like growth on the surface. In the affected area, the lamellae have lost their parallel arrangement, have changed direction, and have a tendency to bend forward into the pedicle. The lamellae are more widely separated and are possibly fewer in the center. The vessels are very numerous, especially near the pedicle. Between the separated lamellae and surrounding the blood vessels, numerous cells of the following types are found: (1) cells, each with a large, pale vesicular nucleus: endothelial and young connective tissue cells; the nucleus is often smaller and distorted by the surrounding tissue; (2) plasma cells; (3) polymorphonuclear leu-

kocytes; (4) occasional eosinophilic leukocytes; (5) small, round cells, apparently lymphocytes; (6) cells in mitosis, most probably of the type mentioned in Group 1. The character and arrangement of the cells varies from place to place. Here the plasma cells prevail, there the polymorphonuclear leukocytes, etc.

The growth projects above the surface of the cornea about 0.93 mm. and is about 2.25 mm. in the greatest meridional diameter. It is button or mushroom-like, overlapping the cornea because it is bigger than the opening in Bowman's membrane, through which it is continuous with the corneal substance. Only in a small area does the growth appear more sloping on the surface and not overhanging. It is not covered by epithelium. On and near the surface, a network of fine fibers, arranged more or less parallel with it, is seen. These fibers have all the appearance of fibrin. Here they enclose an edematous area, there the network contains numerous leukocytes and traces of extravasated blood in other places. The skeleton of the growth is made up of more or less radially arranged fan-rib-like septums of connective tissue and possibly of some remnants of corneal tissue. Small and large vessels often showing bifurcation are very numerous. The vessels are arranged in a fanlike manner as in a papilloma and rather large sinus-like cross sections, filled with blood, are seen near the surface. The space between the connective tissues groundwork and the vessels is filled with cells of various character. They are all of the same nature as those of the affected area of the cornea, but are more numerous and the relative frequency is here different, the cells having large, pale, oval nuclei, and the leukocytes generally prevail. Plasma cells are also more often seen than in the cornea.

Behind the affected area more numerous nuclei are seen than in the balance of the cornea. Some of these are apparently nuclei of lymphoid cells. The posterior surface of the cornea is somewhat bulging toward the anterior chamber and shows undulations. These are due to wrinkling of Descemet's membrane which forms archlike projections into the chamber. There is no break in Descemet's membrane. These arch formations seem to be partly empty; they contain partly fine granular coagulum staining pink with eosin and partly some stratified tissue rich in nuclei and resembling the adjoining cornea.

Over the central part of the cornea, the endothelial cells are almost entirely missing. An occasional one on the posterior surface of Descemet's membrane cannot positively be identified as such and resembles more a lymphoid cell. Only near the angles of the chamber are the endothelial cells forming a continuous layer, but are even here fewer than in the normal eye.

The anterior chamber is about 1 mm. deep in the center. The angle is perfectly free in most sections and the structures surrounding it are almost normal; in a few sections, a peripheral adhesion of the iris to the cornea measuring about 0.4 mm. is seen. The anterior chamber contains only a slight amount of granular, pinkish staining coagulum with a few cells of lymphoid character.

A small focus of lymphoid cell infiltration is found in the iris root and a similar one near the sphincter. A small, broken-up posterior synechia exists at the pupillary margin. Ciliary body, choroid and the other structures show nothing of interest. There is no glaucomatous cup.

From the description, it is quite clear that in Case 2, aside from the characteristic symptoms of corneal burns, not the subject of this paper, such as loosening and desquamation of the conjunctival epithelium, failure of the corneal epithelium to adhere, etc., a condition existed in the cornea which showed all the histologic characteristics of a granuloma, which had developed in the anterior layers and on the surface near the center as a result of the destruction of the corneal tissue by lime. The clinical course of the first case makes it almost certain, in spite of the lacking histologic examination, that here also the growths were pure granulomas. These, though ultimately situated on the cornea, had their beginning in the conjunctiva of the eyeball near the limbus. With the advancing clearing of the necrotic area, the granulomas advanced more toward the center of the cornea, shrinking in the periphery on account of the scar formation at the rear, until ultimately all the remaining granulomatous tissue was choked off by the scar formation and the accompanying reduction of the blood supply.

A search of the literature for cases of true granuloma of the cornea will have to exclude at the outset the formations of granuloma tissue as the result of tuberculosis and leprosy. These cases undoubtedly belong to a different class, as is already evidenced by the etiology, but Bock's cases of granuloma following parenchymatous keratitis, to be mentioned later, cannot positively be classed with these on account of our present views concerning syphilitic parenchymatous keratitis.

TYPES OF GRANULOMAS

If we thus limit ourselves to the pure granulomas of the cornea, it is advisable for practical reasons to divide these into the following three groups:

1. Granuloma in the region of the cornea, the result of a prolapse of the iris following perforation; this might properly be called granuloma of the pseudocornea.
2. Granuloma of the cornea extending from the conjunctiva of the eyeball near the limbus after injuries, etc.: granuloma of the sclerocornea.

3. Granuloma of the cornea, developing away from the limbus and not the result of perforation: granuloma of the cornea proper.

1. It is evident that the cases of our first group cannot be classed as granulomas of the cornea, but they are here considered for purely external reasons only. While not very frequent, they are more often to be observed than the other two forms of granuloma. In our second case, a superficial examination gave one the impression of a granuloma of this kind, such as is depicted by Parsons.¹ In Blaskovics' case, to be mentioned later, this possibility was also considered. In this group belong the two cases of Sichel, his observations 205 and 206, since perforation of the cornea had occurred in both instances. The case of Schmidt-Rimpler was probably of the same kind. His patient, a young laborer, after getting lime in both eyes, had paracentesis done on the right cornea on account of hypopyon. After nine months the right cornea was entirely opaque, everywhere traversed by blood vessels and showing a thick button of granulation tissue.

Schaeche's case of a boy who developed panophthalmitis with perforation, after mortar injury six months before, also belongs here. Large, mushroom-like granulomas formed at the point of perforation.

The case represented by Pagenstecher and Genth on their Plate 12, Figures 3 and 5 may possibly be classed here.

Lawson's case of a cicatrix horn growing from the cornea, seems to be an unusual form of granulation tissue formation developing after corneal perforation.

2. Of the granulomas belonging in Groups 2 and 3 of the foregoing classification, the textbooks make no mention whatsoever. The American Encyclopedia of Ophthalmology contains only meager reference to these conditions on pages 3436 and 5631. A good description by Wintersteiner with enumeration of the cases reported is found in the German Encyclopedia and may be repeated here: "As granulomas of the cornea have been described some few reddish, soft tissue-granulations growing to pea size which later become pedunculated and regressing and developed after an ulcer, an injury, or a chronic inflammation." No mention of the condition is made in the French Encyclopedia.

The only other references to granuloma of the sclerocorneal type in the available American literature are found when Weeks,

1. Parsons: Pathology of the Eye, 1904, 1, 169, Fig. 103.

discussing granuloma pyogenicum of the lids, wonders whether the so-called granuloma of the cornea or of the sclerocorneal margin is not akin to it. The granuloma of the cornea has a tendency to recur, he says. He has examined two sclerocorneal tumors. In the same discussion, Jackson mentions a case of granuloma at the margin of the cornea, in existence six to eight weeks, having a purplish red surface; the edges reminded one of an epithelioma, but were perfectly soft.

In the discussion of burns of the eyeball by lime, Lewin and Guillery mention the following cases in which the granuloma formation began in the conjunctiva of the eyeball and advanced onto the cornea. (1) The cornea of a mason's helper, after mortar had splashed into his left eye, became covered with granulation tissue, reaching from the limbus almost to the center although no ulceration had developed. (2) The case of Desmarres of a mason whose cornea after he had fallen into a lime pit became covered with a sarcomatous growth. (3) After getting lime into the right eye twenty-five days before, a boy showed mulberry excrescences on the swollen conjunctiva and the vascularized limbus. Two months later, the excrescences covered the whole cornea.* After three months, only a small point of the cornea was clear and the remainder was covered by an enormous granulomatous mass pedunculated at the base. (4) Gosart's case of a fleshy pterygium which partially covered the cornea.

In this group also belongs, without doubt, our first case.

Beierle reports a case belonging in this group of hypertrophy of the conjunctiva and granuloma on the otherwise intact cornea of a dog. He assumes injury of the cornea.

3. The most interesting group is formed by those cases in which the granuloma unquestionably originated in the cornea itself, away from the limbus, and did not follow perforation—only resulting from a destruction of corneal tissue, with or without the formation of an ulcer. It is for these cases alone for which the term granuloma of the cornea should be reserved. Search of the available literature discloses only the following cases.

EXAMPLES OF GRANULOMA OF THE CORNEA

Blaskovics' patient, a woman, aged 25, had recurrent eye inflammation with photophobia when a child. When 8 years old, she received an injury to the right eye and a white spot in the center of the cornea resulted from it. Four years before she

came under observation, a red area appeared in the center of the spot and increased gradually. When seen by Blaskovics, the tumor was 10 by 11 mm. in diameter, 1 mm. thick, yellowish red and soft, its surface irregular. Granuloma from an iris prolapse was considered a possibility but tumor of the cornea finally diagnosed. The examination of the enucleated eye revealed no bacteria. The cornea showed absence of the superficial layers in the center. The deeper layers were loosened. Near the growth the cornea was infiltrated with round cells and numerous vessels extended into it from the conjunctiva. Bowman's membrane was absent in the whole extent of the growth. A 1 mm. band of unaffected cornea extended all around the growth. It was divided by connective tissue septums, forming wide meshes. These contained homogeneous intercellular substance with small round cells, and the nuclei were often in mitosis. He considers it a case of typical granuloma of the cornea.

Blaskovics also quotes as true granuloma the three cases of Bock. In his first publication, Bock relates two cases of granuloma formation of the cornea among ninety-two cases of parenchymatous keratitis. The masses disappeared gradually and left dense opacities. His third case was that of a boy, aged 13, who had had parenchymatous keratitis of the right eye some time before and on whose left eye a growth developed during the four weeks previous to consultation. In the center of the opaque cornea, which showed deep vessels, a growth of 3 mm. was visible. It increased rapidly during the two weeks of observation and became constricted at the base. The excised growth showed the picture of a wound granuloma. Its pedicle was covered with one layer of epithelium. The condition is looked on as the result of a circumscribed excessive accumulation of lymphoid cells which after breaking through the lamellae in front of it, could grow unhindered. These cases are not mentioned in Lagrange's excellent work.

The case of Pagenstecher and Genth on Plate 12, Figures 1 and 2, is an undoubted case of pure granuloma of the cornea proper, though some misunderstanding has been created in the literature about it, owing to the use of the word sarcoma in the English translation of the German text. Its granulomatous character is also maintained by Lagrange and by Gayet, whom he quotes.

The granuloma in Raabe's case developed at the seat of a healing septic ulcer of the cornea which had been cauterized. The histologic examination showed granulation tissue.

Hirschberg and Ginsberg describe a large, prominent, coxcomb-like, grayish-red granulomatous tumor on the cornea of a

girl of 9 having tuberculosis. The tumor showed granulation tissue with numerous leukocytes and areas of myxomatous tissue. It was not covered by epithelium.

A vascular pedunculated growth developing rapidly after injury was reported by Bargeton and thought to be possibly a sarcoma, but histologic examination confirmed the clinical diagnosis of botryomycosis. A similar diagnosis was made by Reishaus whose patient, a woman of 46, had much eye inflammation when a child, and whose left cornea showed a small white tumor beginning at the lower margin and extending into the pupillary area. The histologic examination of the excised tumor revealed it possibly to be the product of proliferating inflammation and certain small bodies found are interpreted as "hefa." Axenfeld, reviewing these two cases, says, "There is no proof that any of the cases were any different from what has been described in the literature as granulomas. Further investigation is necessary to determine the pathogenesis of these growths."

Our Case 2 agrees in all histologic essentials with the cases of Blaskovics, Bock, Pagenstecher and Genth, Raabe, Hirschberg and Ginsberg, etc., and is, therefore, one of a pure granuloma of the cornea, of which histologic examination could be made.

Bajardi reports a pedunculated granuloma on the cornea of a rabbit, probably resulting from an ulcer. It was covered with epithelium. Gay found a pea-sized tumor on the cornea of a bitch, consisting of granulation tissue.

It does not seem quite certain that the case of Adler is really a case of granulomas of the cornea although apparently classed as such by Lagrange. Strangely enough, this case does not seem to be reviewed in the contemporaneous German literature. A railroad employee, suffering from gradual diminution of sight with moderate local inflammation of the eyes for two years, showed gray, gelatinous granulations on each cornea, eighteen on the left and fewer on the right eye. The histologic examination revealed them to be a kind of miliary granuloma, consisting of corneal tissue covered by an epithelium much thicker than normal and suggesting those fibrous formations one sees especially on serous membranes following chronic inflammation. But in view of the peculiar histologic findings, the bilateral arrangement, the multiplicity of the growths, and finally because of the occupation of the patient, it might be pardonable to think that the condition of each cornea was nothing else but a kind of

hypertrophy of the corneal epithelium, as seen at times after recurring irritation, produced in this case by repeated foreign body injuries.

A number of cases are recorded in the literature under different names such as fibroma, myxoma, sarcoma, etc., in which the condition described can without undue force be explained as some result of granuloma formation of the cornea either showing greater development of fibrous tissue, or showing changes of a myxomatous nature, etc. Some of them are undoubtedly only changes in a pseudocornea following perforation or destruction of the cornea. As such may be classed the cases of Adler, Benson 1 and 2, Capellini, Falchi, Gallenga, Mitvalsky, Polignani, Qualino and Guaita, Rogman, Rumschwitsch 1 and 2, Scott and Story, Silex, Simon, Szokalski, Zirm, etc. In this connection, Parsons² remarks: "No sharp line can be drawn between purely inflammatory conditions and many so-called growths . . . As to the embryonic spindle-celled type of sarcoma, it may be doubted whether some of them are not simple granulomata (granulation tissue)." Similar opinions are voiced by Lagrange, Blaskovics and Wintersteiner.

It is difficult to say where Stellwag's case is to be placed. The histologic examination, though quite insufficient for classification, is not of such character as to exclude a possibility of the granulomatous origin.

ETIOLOGY

In connection with the question of etiology, one will have to consider two points: (1) Under what condition does granuloma arise in tissues and especially in the cornea? (2) Why did the destruction of corneal substance not lead to any secondary infection but only to the formation of granulomas?

On this point, Mallory says, "Healing by granulation is the repair of wounds where there is more or less loss of tissue which has to be filled in by new formation of cells. The injury is often excessive, much foreign material has to be removed."

It is interesting how well this description applies to all cases of granuloma of the cornea in which a good history of the origin exists. Burns of the cornea by lime or other chemicals will be followed by necrosis and the necrotic material will have to be cast off before repair can set in. The same thing will apply to destruction of the cornea by ulcers, vesicle formation, etc. If the corneal tissue has been destroyed by an inflammatory process,

2. Parsons: Pathology of the Eye, 1904, 1, 264, 265.

such as in parenchymatous keratitis, it need not lead to any loss of substance on the surface, resulting in an ulcer. The process of removal of the necrotic material and the details of repair will be essentially the same, and may, therefore, lead to granuloma formation. It is more difficult to explain how these cases with their long standing necrotic ulceration escape secondary infection and rapid destruction of the remaining cornea. It may possibly have to be ascribed only to a fortunate circumstance that the ever present pathogenic microorganism did not enter the tissues. After vascularization has taken place in the cornea, and even more so after the granulation tissue has begun to develop, the cornea becomes more proof against the attacks of bacteria. In this connection, it is interesting to remember the remarks of Weeks and Axenfeld quoted in the foregoing.

TREATMENT

In the treatment of these cases, it will be good to bear in mind the continued danger of infection and therefore to limit one's self to as little manipulation as possible. Abscission of the granuloma may not be advisable, in view of the several reports of their recurrence, and especially so because such action may open an avenue for infection. It is best to await the absorption of the granuloma in consequence of the scar formation and the accompanying reduction of the blood supply. This plan was carried out in the first case reported.

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DISCUSSION

DR. J. D. BLUM, New Orleans: I have recalled to Dr. Feingold that he saw a case in consultation with me three years ago, a woman of 60 years, who had accidentally spilled carbolic acid, and in about six weeks developed granulation of the cornea, which disappeared under treatment of curretting and the application of tincture of iodine. It was first thought to be a malignant growth, but on taking her to the operating room the condition was improved and I did not operate. Dr. Feingold had forgotten this.

THE CHAIR: Dr. Feingold's papers are very interesting, and very technical.

REPORT OF A CONGENITAL TUMOR OF THE CORNEA OF DOUBTFUL CLASSIFICATION

CAPT. H. H. STARK, M.D., M. C., U. S. A.

EL PASO, TEXAS

Uncomplicated tumors confined to the cornea, either congenital or acquired, are of so rare an occurrence that I feel the case I have recently seen may be worthy of reporting.

History.—A. A., a Mexican girl, aged 15, who was well nourished but of nervous temperament, reported to me on Nov. 6, 1917, having been sent home from school by her teacher with instructions to see a doctor regarding a growth she had on her right eye which she claimed had been there since birth. No family history could be obtained, both parents being dead.

Examination.—On examination the following conditions were found: Both lids were normal in shape and movement. The right eye was slightly smaller and more deeply set in the orbit than the left, having a convergent strabismus of about 20 degrees with no movement to the temporal side past the median line. The conjunctiva and sclera were apparently normal. On the cornea was a growth protruding between the lids with a perfectly round base of about 8 mm. in diameter and about 5 mm. high. It was bluntly conical in shape and slightly flattened, horizontally, from lid pressure. The body of the tumor was light pink, the tip darker in color with faint stippling due possibly to exposure and inflammation. On examination with a loupe the stippling appeared faintly papillary in character but no hairs could be seen. Surrounding the tumor was a distinct area of clear cornea and limbus about 3 mm. wide on the temporal side, shading down to 2 mm. on the nasal side, having what appeared to be a normal curvature. On the nasal side with the aid of a loupe could be seen one large, one medium and several finer vessels passing through the corneal tissue from the limbus, terminating in the tumor, but there was no clouding of the cornea. Through the clear cornea could be seen the iris, normal in color. The pupil was dilated evenly with atropin and with an oblique view showed normal reflex of the fundus. An interview with a relative with whom the patient was living confirmed the history that the tumor had been present since birth. She also said that it had increased in size during the last few years.

Operation.—Photographs of the eye were taken, and a few days later, under a general anesthetic, the growth was removed. On picking up the tumor with forceps it was found to be very hard, requiring a firmer grip than ordinary tissue. After fixing, a cataract knife was passed along the base of the tumor at its

juncture with the cornea, then carefully working and pulling on the tumor thinking there might possibly be a connection some place with the anterior chamber. After loosening part of the base and pulling with considerable force, a distinct line of demarcation could be seen between the tumor and cornea, along which the knife passed, easily peeling the growth from its attachment. About the center of the base of the tumor was a marked bleeding, coming from a point in the cornea, which continued for several minutes after the removal of the growth.

Result.—Examination of the cornea after the operation showed it to have a normal curvature and apparently to be of normal thickness and transparency. There was no sign of scar tissue or injury in any place. The healing was rather slow on account of the large area to be covered with epithelium. The spot in the center corresponding to the bleeding point remained red for at least two weeks as though a blood clot had formed, finally disappearing. At the time of the operation blood was taken for a Wassermann test, which proved negative. After healing, the pupil was dilated with atropin and an indistinct view of the fundus was obtained, but no abnormalities were found. The cornea at the present time shows a thick mottling of scar tissue rather than a dense scar, many places being so clear that a distinct view of the iris can be had. The eye was practically blind, no doubt from the fact that vision had never been developed. Now, seven months after the operation, there is no recurrence. The left eye was normal in appearance and vision.

EXAMINATION OF THE TUMOR

Much to my regret I allowed eagerness on my part to determine the nature of the growth to prompt me to turn the tumor over to our local pathologist with instructions to make frozen sections rather than to have serial sections made. The frozen sections were made from the center of the tumor and stained with hematoxylin-eosin. Examination showed the tumor not to be of ordinary character and some of the sections were sent to different pathologists for their opinions. Later, a part of the tumor was embedded in paraffin, sectioned and stained with van Gieson's stain and sent to the same men, these procedures using about two-thirds of the tumor. The remaining one-third was embedded in paraffin and sectioned serially.

REPORT OF DR. W. W. WAITE, EL PASO, TEXAS

"I wish to report on the pathologic specimen removed from the eye of a Mexican girl. This specimen was a small button or disc-shaped tumor, 8 mm. in diameter and 2 or 3 mm. thick, covered on one side with epithelium, the other side being the raw surface where it was peeled off the eye. The epithelium was roughened in places, containing elevations that might be classed

as small papillae, but for the most part it looked as if it had been subject to considerable ulceration, with an attempt at healing. The whole specimen was hardened in formaldehyd. A small section was taken out through the center for a frozen section. On section the tissue was very firm, and the frozen block cut with difficulty, like cutting a piece of tough leather. Sections stained in hematoxylin and eosin on microscopic examination showed a layer of fibrous tissue, covered by a fairly thick layer of stratified epithelium. The epithelium was somewhat irregular on the surface, and in the space beneath it there was considerable infiltration with small round cells. In none of the sections was there any suggestion of glands or hair. The connective or fibrous



Fig. 1.—Congenital tumor of cornea, front view showing separation of tumor from the limbus.

tissue layer was made up of rather coarse fibers, stained deeply with eosin, in some ways suggesting smooth muscle tissue, but further examination showed there was considerable hyaline change. In the fibrous tissue layer there were small areas of fatty tissue. There was nothing to indicate the origin of these fatty areas. It may be possible that they were the result of a long continued degeneration of the fibrous tissue. The fact that hyaline degeneration was present in the fibrous tissue shows that it was degenerating. To make sure that the fibrous tissue was really connective tissue, sections were stained with van Gieson's connective tissue stain and the entire layer took the red stain. Another piece of tissue was embedded in paraffin and sectioned. The results from these sections were the same as those described in the foregoing. In looking over a large number of sections I

did not see anything that resembled a gland or hair follicle. My diagnosis of this case would be *fibroma*."

REPORT OF DR. F. H. VERHOEFF, BOSTON, MASS.

"The two sections submitted to me show the tumor to consist chiefly of hyaline fibrous tissue. Its surface shows marked papillary outgrowths and is covered with squamous epithelium, beneath which in places there is a moderate infiltration with chronic inflammatory cells. At the base of the tumor there is an abundance of adipose tissues. Diagnosis: *dermoid tumor of the cornea*.

"The unusual feature of this case was the marked papillary character of the surface, and the fact, apparently indicated by the photograph of the patient, that the tumor was not connected with the limbus but was confined to the cornea. There are no sweat or sebaceous glands in the section, but these are not necessary for the diagnosis. It is possible, however, that other sections may show such glands. I have seen one similar case, in which in addition there was an independent teratoid tumor of the bulbar conjunctiva."

In a subsequent letter, he states: "The large amount of fat tissue present in the other sections you sent me rules out a simple fibroma. You could call this tumor a *dermo-fibro-lipoma* if you wished, but it is essentially of the same nature as a dermoid."

REPORT OF DR. ALLEN J. SMITH, PATHOLOGICAL DEPARTMENT, UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA

"Through the kindness of Dr. William Zentmeyer, sections of this tumor were submitted to Dr. Allen J. Smith, Pathological Department, University of Pennsylvania, who writes: "I must confess to an inability to recognize the tissue as corneal had I not been told of its origin. The epithelium is too thick, forms penetrating roots or pegs, and in its best places is not flat enough. I cannot distinguish a Bowman's membrane; the matrix is not lamellated, is too cellular, and in its vessels (of course because of the fact the sections do not reach all the way through the cornea), I find no Descemet's membrane in its lining cells. True, too, in several of the sections, fat cells as in isolated cells occur in the matrix. The cornea apparently had small nodosities or granular projections on its surface, as small fibrillar protrusions appear in the section covered by epithelium. The thickened epithelial layer, such nodosities, even the epithelium penetrations, and of course the fibrous tissue, especially the finer parts close to the surface, might well be explained as due to the vascularization referred to, on the basis of a chronic inflammatory process, but I can scarcely think of the adipose cells in this relation. I feel this feature can only be explained by the suggestion you give me that the condition is a dermoid. With this idea that I have skin instead of, or added to, corneal tissue, the

thickening of the epithelium, the nonlamellated fibrous matrix, the presence of blood vessels, and particularly the presence of fat, are all in conformity, but I wish Dr. Stark could also find a hair or two or a skin gland in some of the material to complete the picture. I am inclined to agree very strongly that this is a dermoid, but I would urge further search for one of these structures as final proof, although I do not feel that failure to find one or the other bars the diagnosis, which stands more as the most probable condition at any rate. I feel that a chronic inflammation has, however, coexisted, the swollen epithelial cells, the epithelial hyperplasia in producing the penetrating roots,



Fig. 2.—Tumor of cornea from the side showing protrusion of tumor.

the turgid capillaries and the cellular investments along these and some of the older vessels, and the dense fibrous tissue areas met with, I think, argues for this, and doubtless this has occasioned a little nodular papilla as well. I do not seriously question the diagnosis of a dermoid, but feel that he may be able to establish the point beyond a shadow of a doubt if he will continue the examination of the material."

After receiving these reports, serial sections of the remaining one-third of the tumor were made and carefully examined, all showing the same character of tissue as was found in the other part of the tumor. No sign of hair follicles or glands were found, nor do I believe there were any in the other sections."

CORNEAL TUMORS IN GENERAL

Tumors of the cornea are divided as other tumors into malignant and benign, the malignant being sarcoma and epithelioma; the benign being fibromyxoma, papilloma, dermoids and teratoids.

In considering corneal tumors, Parsons¹ states:

"A review of the recorded cases of corneal tumors leaves one in great doubt as to their true status. No sharp line can be drawn between purely inflammatory conditions and many so-called growths. There is an even gradation from the simple corneal scar to the myxoma on one hand and the papilloma on the other. . . . The same applies with equal force to the new growths both benign and malignant, for all are vascular, though in varying degrees, and derive their vessels from the limbus. Most doubtful of all are the malignant growths, especially the epitheliomata and the endothelial type of sarcomata."

He is justified in his contention that growths of any kind confined to the cornea are rare, as it is a well known fact the favorite seat of practically all tumors of the eye, with the exception of a lipoma, is either astride the limbus or connected with the limbus in some way. This fact becomes more evident when one makes a search of the literature. In reviewing different articles on tumors of the eye, it was found difficult in many cases to determine from the description whether the tumor was confined to the cornea or had some connection with the limbus. It is true that only a slight connection with the limbus is necessary, for as the growth increases in size, advancement is usually made over the corneal surface. Many cases of reported corneal tumors are found, on careful reading of the articles, to have connection with the limbus. Even many of those looking like pure corneal growths are questionable and should not be so classified. However, including doubtful cases, the number of tumors confined to the cornea is very small, in comparison with all tumors of the eye.

A fairly good search of the literature was made, classifying the tumors under the head of malignant growth, including sarcoma and carcinoma, and benign, including papilloma, fibromyxoma, dermoids and teratomas, the equivalent being as follows: malignant growths, twenty; papilloma, four; fibromyxoma, one. Of dermoids and teratomas there were none unless we include multiple tumors, where one was situated on the cornea, the second on some other part of the eye. Even these are very rare. Just why there are so many more malignant than benign growths is

1. Parsons, J. H.: *The Pathology of the Eye*, New York, G. P. Putnam's sons, vol. 1, p. 264.

a doubtful question. Two views can be taken on this point: (1) that many of the malignant growths are simply degeneration of a former benign tumor, and (2) that the tendency is stronger with the oculist to report a malignant growth than one of a benign character. No doubt both of these views have a bearing on the question.

In considering the tumor, we can safely exclude malignancy. Of the benign tumors, the history of the case and the histologic structure of the growth would exclude papilloma, leaving us only the dermoid and fibroid to be considered.

DERMOID TUMORS AND THEIR ORIGIN

Parsons² has made the following statement: "Dermoids are lenticular yellow or reddish congenital tumors, which occur



Fig. 3.—Eye after removal of tumor. Front view.

astride the corneal margin usually on the outer side, containing the elements of skin (stratified epithelium, hairs, sebaceous glands, etc.). They were described as early as 1742, and four cases were published by Wardrop in 1808. They were named 'dermoids' by Ryba in 1853. While commonest at the outer part, they occur rarely at any part of the limbus, even under the outer canthus. They are often associated with other congenital malformations (according to Picque 27 out of 94) such as colobomata of the lids, fleshy bands from the globe to the face, etc."

Various theories have been suggested for the explanation of dermoids. Ryba suggested failure of complete closure of the lids, with consequent cornification of the conjunctiva, and cited the correspondence of dermoids and lid-notch. This view is

2. Parsons, J. H.: *The Pathology of the Eye*, vol. 1, p. 132.

accepted by Bland-Sutton and Treacher Collins; the latter regards cryptophthalmia, in which the whole surface of the eyeball is covered with skin, as the ultimate manifestation of dermoid development. Gallenga drew attention to the plica semilunaris, which in fetal life, covers the globe like its phylogenetic equivalent nictitating membrane. He thought it might remain adherent to the membrane. Osborne thought the tumor might be a remnant of the epiblast which forms the lens. Van Duyse considered it was due to an adhesion of the amnion to the eye. While it is not the province of this paper to discuss the origin of these growths, it is worth considering as it may be possible that both the theory of Ryba and that of van Duyse are correct, and that those showing true dermal character come from the lid, while those without the dermal characteristics come from the amnion.

CLASSIFICATION OF DERMOID TUMORS

On the classification of these tumors many interesting and instructive papers have been written. Among the early ones is Gallenga,³ who classifies them as: (1) pure dermoids; (2) lipodermoids, and (3) mixed form or lipomata containing acinous or rather acino-tubular glands, or cartilage. Concerning the origin of the pure typical dermoids, Gallenga accepts Ryba's explanation, according to which dermoids develop as the result of disturbed closure of the oblique facial fissures, and thereby also of the palpebral gap.

Saemisch⁴ states that:

"Dermoid of the conjunctiva is the designation usually applied to tumors which are congenital and closely related to the skin in their histologic feature. They are accordingly to be interpreted as teratoid tumors. However, the histologic components of the skin may be unequally developed in individual cases, the epidermoidal structures of the skin, the hairs as well as the glands (sebaceous and sweat glands) being much more distinctly manifested in some cases than in others. The degree of development of the subcutaneous fatty cellular tissue is especially variable, being sometimes merely suggested, whereas, on the other hand, it may form the main bulk of the tumor. Nerves, vessels, and smooth muscle fibers are likewise seen variably developed in their growths; more rarely discs of cartilage have been noted."

Notwithstanding the great variability in the histologic behavior of these tumors, they share the common peculiarity of representing cutaneous structures which have been developed in the

3. Gallenga, C.: *Ann. d'ocul.*, Brussels, 1885, 95, 215.

4. Saemisch, Th.: *Graefe-Saemisch Handbuch des gesamten Augenheilkunde*, vol. 5, part 1, p. 604.

conjunctiva, although the individual tissue varieties of cutaneous character are by no means necessarily developed to the same degree. Hence it does not appear to be justifiable to select this factor as the criterion for a classification of these tumors, as suggested by Gallenga, but it would seem more appropriate to have the classification of these tumors on their topography.

Fuchs⁵ says:

"Dermoid tumors are flat growths of a solid consistence, which, so to speak, straddle the margin of the cornea, being situated partly in the conjunctiva and partly in the cornea, with which latter they are immovably connected. They most frequently occur on the external (temporal) side of the cornea. Histologic examination shows that the growth possesses the composition of the external skin. It consists of a stroma of con-



Fig. 4.—Eye after removal of tumor. Lateral view.

nective tissue, covered with epidermis and contains hairy follicles and various glands. It is, so to speak, an island of skin on the surface of the ball."

The latest and perhaps most exhaustive paper on dermoids is that by Pignatari,⁶ as follows:

As shown by numerous reports in the literature concerning dermoids of the corneo-scleral junction, there is not one constant structure for these mixed teratomata. According to Gallenga's classification, which is adopted by the author, these growths are divided into several groups, as follows:

A dermoid of the conjunctiva is a congenital tumor whose histologic structure has very close relations with that of the normal skin. Its volume is usually small, seldom more than half a nut; the color is usually whitish, often slightly pink or yellowish,

5. Fuchs, E.: *Text Book of Ophthalmology*, English Translation, 1911, p. 209.

6. Pignatari, G.: *Monograph*, Turin, 1913, *Review and Ophthalmic Review*, 1915, 34, 57.

a tint attributed formerly to the presence of fat, though this has been shown not to be the case. The form most common is a truncated cone; but hemispheric is not uncommon, and occasionally they may be pedunculated. The surface is sometimes smooth, sometimes slightly irregular; rarely it presents a series of small irregularities arranged similarly to those of the skin. Sometimes, the surface of the tumor may become covered with fine drops of fluid while it is being examined; these drops come from small glands in the subjacent tissue, and their ducts may be seen by close inspection with the aid of a powerful loupe. The surface usually bears hairs, varying in length with the age of the patient. It is rare to see in children anything but a fine down. The site of the tumor is important: commonly they ride the lower outer part of the sclero-corneal junction, more rarely they are purely conjunctival; a completely corneal seat is the least common. In four instances only has the seat been the caruncle. The histology of these tumors shows their close relation to the skin; all the parts of the true skin are to be found; hair glands and sebaceous glands are almost constant; on the other hand, sweat glands are comparatively rare, though not so infrequent as they were held to be by the earlier observers.

Under the name of lipodermoids, Pignatari includes those forms which are congenital and have a superficial layer like that of dermoids, but which differ in containing a large quantity of fat, as well as certain other elements, acinous glands and smooth muscle fibers, which are less frequent in this form. The normal situation of a lipodermoid, in which some 70 per cent. are found, is between the insertions of the rectus superior and the rectus externus. The structure of these lipodermoids varies within wide limits; sometimes the dermal features predominate, sometimes the fat; often there will be so little development of the cutaneous elements that they may be overlooked, unless series of sections are taken of the whole mass. In a considerable number of cases a mass of cartilage will be found in the deeper part.

The pure conjunctival lipoma is a tumor developed in the subconjunctival tissue and devoid of all cutaneous element. Many of the cases which appear to be lipomata on clinical examination, prove to be lipodermoid when subjected to the microscope. Great difference of opinion still prevails among pathologists as to the constitution of lipomas, and as to their congenital origin.

MIXED TERATOMAS

As far back as 1888, Gallenga suggested this third division for these tumors, and placed in it those cases in which lipomas



Fig. 5.—Section of tumor showing connective tissue masses and fat cells in Stark's case of tumor of the cornea.

were found to contain acinous or acino-tubular glands, or remains of the semilunar cartilage. Later, Gallenga redivided this group into two: (1) mixed teratoma with mucous surface, and (2) mixed teratoma with cutaneous surface. The origin of the mixed teratomas is probably different from that of the tumors so far considered. They contain acino-tubular glands, and these glands often very closely resemble the lacrimal in structure. Gallenga and some others are inclined to consider them really derived from the lacrimal gland and to be simple ectopia glandis; they reason thus from the fact that the secretion from the gland is neither wanting nor abnormal. Gallenga imagines this also that the growth of this aberrant gland accounts in some cases for the presence of cartilage or bone, those fragments having been cut off from the orbital margin by the gland in its extension. Probably, however, he thinks in most cases the cartilage is derived from the cartilage in the membrana nictitans, which has become in the course of development adherent to the eye.

Concerning the development of teratomas, Pignatari accepts Gallenga's views in accordance with which the typical dermoid is a part of the lid margin, which has become adherent to the globe, and has gradually become separated from the lids by the development of abnormal lines of division, which form furrows growing gradually deeper until the dermoid is cut off from the lid and remains a part of the globe. The other forms of teratoma are due, in his opinion, to ectopia of some part of the lacrimal gland, which takes up a new position because of the faulty discretion of some of its parts. The presence of amniotic bands is only a rare cause.

A comparatively large proportion of dermoids are associated with some faulty development of the eyelids or face. Presumably, coloboma of the lid is chiefly due to the adhesion of a part of the lid-margin to the eye and its separation afterward; thus a gap is left in the lid, the coloboma, which is accurately filled by the dermoid.

FIBROID TUMORS

The idea prevailing with practically all authors is that fibroid tumors originate from scar tissue, most of them being simply hyperplasia of this tissue. This is agreed to by Parsons,⁷ but he says there are cases which are apparently true fibromas. Regarding the structure, he says, "they consist of irregular bands of fibrous tissue, with scant flat and round cells."

7. Parsons, J. H.: *The Pathology of the Eye*, vol. 1, p. 257.



Fig. 6.—Section of tumor of cornea (Stak's case) showing papillae on part of the surface.

CONCLUSIONS

After reading the literature on corneal tumor and considering this case I have come to the following conclusions:

1. Corneal tumors occur less frequently than the literature would indicate.
2. In order to be classified as a corneal tumor the growth must have no connection at any place with the limbus.
3. That dermoid may be used as a term to classify congenital tumors, even those of simple histological structure containing no dermal tissue, hair or sweat glands.

DELIMITING KERATOTOMY IN THE TREATMENT OF SEVERE CORNEAL ULCERS

H. GIFFORD, M.D.

OMAHA

Some twenty years ago, in a case of bad corneal ulcer, in which on account of the proximity of the iris to the cornea, I was unable to make the classic Guthrie-Saemisch incision, I made a cut along the central border of the ulcer and observed that the infection while it spread along the cut did not cross it. This experience led me to systematically make this form of incision in ulcers where a very slight progress would mean a serious loss of sight. I have found that, as a rule when other suitable measures are used, the ulcer seldom crosses the cut, although it sometimes spreads around its ends.

TECHNIC OF AUTHOR'S METHOD

I aim to make the incision as near as possible to the infected area without quite touching it, the cut thus describing a near tangent to the border of the ulcer (Fig. 1). In hypopyon keratitis, I do not rely on this cut alone, but before making it I make a thorough application of optochin, if the case is a pneumococcus one, or an application of 20 per cent. sulphate of zinc if it is of Morax bacillus or of doubtful origin. In addition, as soon as the chamber is filled up, either the same day or the next day, I burn through the center of the ulcer with the galvanocautery; and, unless this opening leaks sufficiently to keep the chamber empty, I reopen it twice a day and continue such other applications to the ulcer as may seem best. The delimiting cut seldom needs to be reopened; but if the infection should spread across it, it may have to be repeated farther on or if the infection does not cross the cut but tends to spread along it, other delimiting cuts at an oblique angle to the original cut may be made to check the progress of the infection in other directions (Fig. 2). The cut is best made with a very sharp, ground down cataract knife and should be made nearly as long as the greatest diameter of the ulcer.

A PREVENTIVE OF SPREAD OF INFECTION

It will be noted that this form of cut is not recommended as a cure for the ulceration but merely as a barrier to gain time and prevent further loss of sight until the other measures can

stop the infection; just as a hastily constructed trench may hold the enemy until the big guns can be brought up to pulverize him. It is, therefore, not recommended as a substitute for the Guthrie-Saemisch cut but as an auxiliary to it or to whatever measure for keeping the chamber open one may prefer. In most cases I prefer to open the chamber with the cautery because with the full-sized Guthrie-Saemisch cut the iris almost invariably becomes adherent to it.

MODE OF ACTION

In searching for an explanation of the efficiency of the delimiting cut one at once encounters the peculiar manner in which corneal wounds heal. It is well known that instead of healing by the immediate interposition of fibrin, such cuts heal through the formation of an epithelial plug. We all know with what rapidity corneal epithelium regenerates itself and it is not surprising to find that a corneal cut is completely filled within a very few hours with a wall of epithelial cells which only very

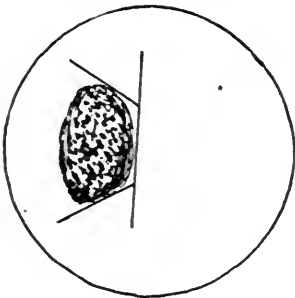


Fig. 1.

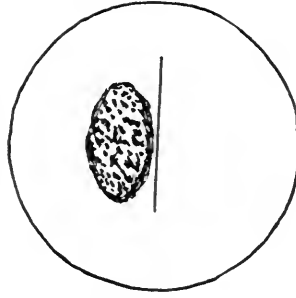


Fig. 2.

Fig. 1.—Simple delimiting keratotomy.
Fig. 2.—Delimiting keratotomy with auxiliary incisions.

gradually are displaced by the regenerating corneal tissue. That such an epithelial plug might offer more resistance than the corneal fibers to an encroaching infection might be inferred from the fact that nearly all the severe ulcers progress by undermining the epithelium instead of attacking it directly.

COMMENT

Although I have used the delimiting keratotomy most frequently in hypopyon keratitis, it is equally useful in any form of rapidly, or obstinately, spreading ulcer. I have used it to advantage in a few ulcers of the rodent type, also in cases of ring ulcer. In one case of the latter, I am quite certain that I saved the patient's sight by making cuts across the advancing ends of the ulcer. In cases of this sort when the cuts are made close to the

margin of the cornea, it is not necessary to go through the entire thickness.

Although I have practiced this cut for many years, it is only recently, on looking up the literature, that I found a similar cut had been recommended a number of years before I used it. The evidence of this I found in Beard's "Ophthalmic Surgery" (1914, p. 392) where it is stated in discussing the Saemisch cut that "Alfred Graefe and Meyhofer taught to make the cut as a tangent to the ulcer and thus slit the infiltrated border and that in the direction of its greatest progress." As I have not been able to find the original article of these men, I can give no details of their theories or practice.

DISCUSSION

DR. JOHN FOSTER, Denver: Any means of helping us in these destructive ulcers of the cornea is received with interest by all of us. From the simple fact that so many different methods have been devised we may see how often they are unsuccessful, even in the hands of the best of us. The Guthrie incision has been unsatisfactory in many conditions. Whether the direct cut has been the healing measure, or the relief of pressure on the cornea is the healing element, has been in doubt many years. The delimiting cut outside the ulcer is on the same line as Meyhofer, and even von Graefe proposed to cut through the healthy tissue. Anything that will help in these cases will be hailed with interest. An ulcer of this description often tends to take a certain direction, and this method will cut it off as we try to cut off an infection in the skin with iodine, etc.

Not all of us would bring these things to the attention of the Society, but to give the profession the benefit of anything of this sort would be a help oftentimes. Every man in this room has some little peculiarity of operative technic, and he goes on through years and years and says nothing about it, and I think the profession is too modest. All the men in this Society are well posted on the ordinary technic, but some little specialty of this kind we should have our attention called to. I hope this will be an encouragement to others to report on their successes.

DR. JOHN GREEN, JR., St. Louis: I have come to rely greatly on the method of treating pneumococcus ulcer of the cornea devised by Shahan of St. Louis. Many of you heard his papers in Detroit and New York. The method consists in the application to the ulcer of a fixed quantity of heat. A hollow metal tube containing a thermometer is held in a spirit lamp until 165 F. is registered. This is then put in an insulated jacket. When the thermometer registers 158 F. the tip is applied directly to the ulcer and held in contact therewith for one minute. Shahan has proposed this method for pneumococcus ulcers only and we in St. Louis have used it only in pneumococcus ulcers, but it may be efficacious in ulcers of other origin. The effect is little short of magical. I have had great success with it, and I know that Shahan has a series of about fifty cases, all of which have been successful, in the sense that the infective process has been definitely stopped by one or two applications. The effect of the application is to limit the process of infection. I have abandoned all other forms of treatment, as nothing that I have ever tried is nearly as efficacious as the thermophore. You do not kill good tissue. You merely heat it to the point at which the pneumococcus is killed. You do not destroy the epithelium, which is merely whitened for a little area surrounding the ulcer. Try the method and be convinced.

DR. H. A. SMITH, Delta, Colo.: If you heat it only to 168 F. and then let it drop down it will soon cool, but if you heat the whole instrument you will maintain it for the whole minute; it will otherwise drop 30 or 40 degrees in a minute. You should have the whole instrument where it will be warm. The improvement begins at once and in ten days the eye is clean and your patient goes home. I have been thoroughly impressed, and I have had eighteen or twenty cases. With one exception the results have been as striking as have been mentioned. In one case I used nothing but boric acid to see if anything else did it. The healing was but slightly delayed.

DR. H. B. YOUNG, Burlington, Iowa: Several years ago I began employing a method, which I have not seen mentioned beyond my brief report to the Chicago Ophthalmological Society, in place of the Saemisch section for these burrowing ulcers with small external openings. (Illustration on board.) It is a crucial incision into but not through the membrana propria, and extending well into the clear marginal tissue. The four flaps are pushed up in turn by a small cotton shod applicator just moistened with phenol, and the whole area scrubbed; and particularly at the periphery into apparently clear tissue. The flaps are replaced, and I have seen prompt relief from pain and the minimum of scar.

DR. R. H. T. MANN, Texarkana, Ark.: Dr. Gifford has upset my ideas of treating ulcer. In making his cut, instead of extending it in this direction (illustrating), I try to preserve as much of the area of the cornea as possible to prevent the infection in another part of the cornea where there had been an abrasion or wound of any kind. Is there not more likelihood of the center of the cornea becoming infected, after an incision has been made in the center of the cornea of an eye in which infection already exists, than if the incision is made in the corneal ulcer? (illustrating). I want to know if it is not probable that a larger area will thus become infected.

DR. HAROLD BAILEY, Springfield, Mo.: I was greatly interested in Dr. Gifford's paper. This class of corneal ulcers have always given me a great deal of trouble for the reason that no two of them act exactly alike. I was formerly afraid to make the Saemisch incision for fear of infection or iris prolapse. Of late years I have learned to regard it as one of my best friends in time of greatest need. A microscopic section of these ulcers often shows small burrows or pockets of infection spreading out from the ulcer under the corneal epithelium, and even in the deeper layers. Here, I believe, the intraocular tension often closes the mouth of the pocket and retards in a measure the drainage. (Illustrates.) The Saemisch incision, of course, reduces this tension as does the incision mentioned by Dr. Gifford. It seems quite possible that the location of the incision is not of great importance as regards the checking of the ulceration. Probably any incision by reducing the intraocular tension will give the same result.

DR. GIFFORD (closing discussion): As to the question Dr. Mann asked, as to the risk of infection of new tissues by making this cut, I can only say that the fact remains that such infection does not occur, but the infection stops along the cut. It will follow along one side and the other side will be perfectly clear.

Dr. Young's method of splitting the edges of the ulcer is good. In case of serpent ulcer where you have an overhanging edge, I have slit the margin in this way and then cut off what I could.

This thermophore must be a wonderful thing and I reproach myself for not having secured one. Where do you get them?

DR. GREEN: From Dr. Shahan.

THE REFITTING OF DISABLED SOLDIERS AND SAILORS *

CASEY A. WOOD, M.D.

Lieutenant-Colonel, M. C., U. S. Army

WASHINGTON, D. C.

We all know what happened to the soldier and sailor disabled in our former wars. After a few brief weeks of hero worship, marked, perhaps, by a kindly tolerance of his oft-told tales of adventure in the Army and on the battlefield, he was given a pension and in some old soldiers' home or as a dependent on the semicharity of neighbors or relatives, eked out a half-forgotten existence, a bore to himself and useless to his country. It is true that a few of these returned invalids, in spite of their handicaps, resumed their former occupation or adopted some new industrial activity, but their number was so small as to be almost negligible.

As a matter of fact, the obstacles that lie in the way of a resumption of industry after the combatant has passed weary months in the field and in various hospitals, are to the sick or wounded man almost insurmountable. Unless prompt and continuous help is given, he lapses into a state of discouragement from which it is difficult to arouse him.

It has long been recognized that to be most effectual this mental and moral assistance, this "cheer-up," this achievement of victory over wounds, should begin at the earliest possible moment. It should be pre-arranged and definitely organized. All the belligerent countries have attacked the problem from this viewpoint and by conducting, chiefly through nurses and medical officers, a campaign of education, early teach the disabled man that his condition is not as hopeless as he probably thinks it is, and that the Surgeon-General and other governmental agencies will see to it that his disablement will be reduced to the minimum, that he will be refitted for industry, and that his family and he will be supported while he is undergoing reconstruction. He will be further assured that his war risk insurance compensation for disability will not be reduced as a result of his increased powers through reconstruction. Finally, a desirable job will be found that will enable him to become an independent, wage-earning citizen, taking part in and enjoying the daily life of his own community.

* Approved for publication by the Board of Publication, Surgeon-General's Office.

This verbal campaign is further strengthened by posters displayed in the hospitals and on board ships returning from overseas, by cards entitled, "What every disabled soldier and sailor should know," by booklets telling how, when and where physical and vocational restoration is accomplished and, last but not least, by moving pictures, such as I expect to exhibit to you this evening.

In these and other ways we spread the good news that there are really no longer any "cripples," but only a readjustment of the soldier's reduced powers to altered conditions and that the so-called disabled man can, if he wishes, improve and apply these powers, that he may even adopt a trade or profession more congenial or more lucrative—or both—than he followed before his injury or illness.

Who, let us now ask, are the war-disabled requiring rehabilitation?

Naturally one thinks of those who have lost eyesight or hearing or a limb—and these cases are of great importance and require special attention—but they really make up only about one-third of the whole number of the handicapped. We must not forget the tuberculous, constituting a large percentage of the total, the nervous patients, including the so-called "shell-shock" cases, the insane (curable and incurable), those with serious diseases of the heart, those that have been "gassed," those with rheumatism, and many with other internal affections. Any of these may suffer from a handicap even worse than the loss of a leg or an arm.

CAUSES OF DISABLEMENT

An approximate idea of the causes of disablement in the service may be obtained from a table based on statistics of the British Army and Navy during the present war. The figures indicate the number of cases in every thousand of disabled men, excluding officers:

| | |
|--|-----|
| Injury to eyes..... | 32 |
| Amputation of leg..... | 30 |
| Amputation of arm..... | 19 |
| Injury to leg not requiring amputation..... | 121 |
| Injury to arm not requiring amputation..... | 82 |
| Injury to hand not requiring amputation of whole hand..... | 61 |
| Injury to head (including six or seven cases of paralysis) | 47 |
| Hernia | 7 |
| Miscellaneous wounds and injuries (including about five cases of paralysis)..... | 53 |
| Total wounds and injuries..... | 453 |

| | |
|--|-------------|
| Diseases of chest (including, say, 60 tuberculous cases) | 124 |
| Rheumatism | 50 |
| Diseases of heart..... | 110 |
| Epilepsy | 11 |
| Nervous cases | 47 |
| Insanity | 9 |
| Deafness (partial, or, in comparatively rare cases total) | 26 |
| Frostbite | 10 |
| Miscellaneous diseases (among which may be noted, besides a few cases of paralysis, Bright's disease, ulcer of stomach, debility, and varicose veins)..... | 160 |
| Total cases of disease..... | 547 |
| Total wounds and injuries..... | 453 |
| | <hr/> 1,000 |

It may be added here that the number of men requiring extended treatment and vocational training constitute a relatively small proportion of the total number of those discharged from the Army or Navy. Although soldiers and sailors suffering from certain chronic diseases, such as tuberculosis, insanity and other mental and nervous disorders may require treatment for a considerable period under special conditions, yet the time of retention in Army hospitals after return to the United States will be relatively short. Some will be so nearly well as to permit of their discharge almost immediately on landing. A considerably larger number will require treatment for a week or even a month or two in the general hospitals. About 90 per cent. of the returned disabled men will be discharged after a comparatively brief period of treatment and will require no special measures of physical or vocational reconstruction.

Our government has already provided ample hospital accommodation for all these injuries and diseases and hundreds of soldiers are now being restored to health through the agency of the Surgeon-General and his medical officers, and receiving meantime, physiotherapeutic, including occupational and other forms of prevocational training.

When the soldier or sailor has been as far as possible restored to health and is incapable of further military service, he is discharged from the Army or Navy and transferred to the governmental civilian agencies, especially the Federal Bureau for Vocational Education, appointed by Congress for the completion of his education or reeducation.

It is estimated that the proportion of the total number of disablements calling for reeducation for an occupation different from the one formerly followed by the disabled combatant is

about 10 per cent. In other words, under the care of the Surgeon-General will fall about 90 per cent. of returned invalids requiring reconstruction, including those who will also, under him, complete their occupational career. When discharged from the Army cured, as nearly as may be, of their injuries or disease, most of these are expected to pass directly into industry, following their old or a new occupation.

OCCUPATIONS OF DISABLED SOLDIERS

The task of finding a suitable job for returned invalided soldiers and sailors, of exercising supervision over them after their return to work, will devolve on the Federal Board for Vocational Education, on the War Risk Bureau and probably, also, on the American Red Cross.

As has been pointed out by Mr. Lakeman of the American Red Cross and others:

The reconstruction of the disabled soldier is in the first place a matter of public duty which is called for by every consideration of national gratitude and justice. This alone is a sufficient reason for the fullest expenditure of needed effort and funds. But also sound national economic policy requires that the state, in order to conserve industrial and labor resources, should make skilled workmen of its disabled soldiers and find them employment. After this war every soldier who returns to a condition of idle dependence on government support will help to diminish the productive power of the Nation. With the terrible wastage of men and materials every ounce of constructive energy will be needed to repair the damage and to face the renewed intensity of competition in trade and industry. In this country the channels of immigration are closed during the war and the draft on the labor market is unprecedented. The supply of skilled labor is no longer inexhaustible. In the field of industry, as in that of natural resources, the era of waste and extravagance is over and every element of productive man power must be conserved as an indispensable national asset.

There is, however, another argument in favor of recalling the disabled man to a useful life. If the handicapped soldier or sailor for any reason fails to make sure his industrial calling—fails to take advantage of his opportunities—he becomes a part of the nation's human scrap heap, an unhappy victim of his own and, perhaps of his family's, indolence and neglect. It is the salvation of a soul as well as of a body for which the rehabilitation activities are striving and for the accomplishment of which we ask your assistance.

The disabled soldier's decision as to his future must be backed up by the unhesitating support of his community. No system of

governmental or private vocational education can succeed unless the man's will to succeed is stimulated and his ambition aroused by a well developed public opinion. No government bureau can exercise authority over the man's personal will to make or neglect his future. For example, it is easy to imagine what a powerful influence the supporting opinion of the family and of the neighbors may be in inducing a man to forego the false glitter of an immediate job in favor of the sound metal of thorough training for an independent future. To convert the man to the wiser view it will sometimes be necessary to convert his family first; certainly his friends, relatives and companions must believe in the desirability of this victory over wounds and this will to conquer difficulties if the disabled man is to be successful in the contest with the power of darkness.

DEAFNESS AND BLINDNESS

It is not possible in one evening to enter into details of the therapeutic care and training now being given to even the most important disabilities of our returned invalids, but I would like to say something more about the war deaf and the blind.

The blinded warrior makes a strong appeal to the emotions, although, perhaps for this very reason, the extent and importance of this form of disability has been exaggerated in the public mind. The French experience indicates that only a half of 1 per cent. of the soldiers engaged in battle are blinded, the reason being, probably, that injuries sufficient to make a man blind in both eyes generally prove fatal.

Some of the foremost educators of the blind have cooperated with our government in devising, perhaps, the most complete system of reeducation and after-care of blinded soldiers which has ever been undertaken in any country. Based on careful study of the best methods of foreign countries, and supported with generous appropriations and ample private contributions, which make possible the most complete equipment, this plan will follow the soldier through all the phases of reeducation, find him employment or a market for his products, and insure sympathetic and intelligent cooperation from his family and from the community.

To carry out this program a fortunate combination of public and private resources has been effected. Through the generosity of Mrs. T. Harrison Garrett, a suitable estate of 99 acres at Baltimore was offered to the Surgeon-General and accepted as the central hospital and school for blinded officers and men. The

Red Cross War Council has supplemented the Army's plan by creating the Red Cross Institute for the Blind, in order that certain phases of the social and economic supervision of these men may be provided for. The medical officer of the Surgeon-General's staff, who is responsible for the official Army program for the blind, has also been made director of the Red Cross Institute for the Blind, and will work in close touch with the Federal Board for Vocational Education, thus insuring thoroughgoing unity of policy and control.

This combination of an official organization with additional private resources will make possible the continuous and perpetual care of blinded soldiers after they are discharged from the Army. Not only education and placement are contemplated, but subsequent industrial supervision for the balance of the soldier's life. It must be remembered that many blinded soldiers will, in addition, be suffering from other forms of disability. In blindness caused in the industries, 98 per cent. of the eye injuries are without such complications, whereas in war, 40 per cent. are multiple, that is, these blind men also have amputations of arms or legs or both, facial burns, destruction of jaw, and so on. To these particularly unfortunate individuals, the nation owes a lasting duty. Patriotic zeal will provide adequate care for these men at present, but only invested capital and far-reaching plans will make their future certain.

GENERAL PLAN OF THE WORK

As soon as American soldiers blinded in battle recover from their immediate wounds at the base hospital, the special work for them will be commenced. At first the chief effort will be to amuse them and inspire them with some degree of hope. Later, as the men improve, they will be concentrated at the port of embarkation in France under care of a special staff which will expand the measures of elementary instruction, teaching the men to get about and care for their immediate needs, in other words, teaching them "how to be blind." On the way home they will be grouped according to the degree of injury, and on arrival in the United States they will be sent to United States Military General Hospital No. 7 at Baltimore for further medical and surgical treatment and special teaching. The technical training given by the government will be the very best obtainable and only paid professional instructors will be permitted to give it. The idea of the government will be to place every blind man in a condition to take care of himself and those dependent on him.

Where it is possible for a soldier to return to his old occupation or one allied to it, the aim will be to have him do so. Otherwise a special suitable occupation will be taught and in many cases it is hoped that the man will be able to command a larger salary after taking the training than before he lost his sight. Among the trades and professions which will be taught are broom-making, mattress-making, rug and carpet weaving, telephone operation, farm work, mat-making, tuning in piano factories, winding of coils for armatures, piano tuning as an individual trade, salesmanship, massage, typewriting and dictaphone operating. There will also be ample facilities for recreation in the school, including table games, dancing, singing, cross-country walking, roller skating, attendance at theaters, musicals, and other entertainments, medicine ball, push ball, punching bag, relay races, gymnastics, field sports, running, climbing, swimming, diving, and skating.

Some of the most interesting features of the general plan will be made possible by the supplemental Red Cross appropriation. The Red Cross Institute, for example, will undertake to provide a home and transportation to Baltimore for the relative who will be responsible for the care of the blinded man when returned to his home. It is the intention to instruct one such relative in the government training school side by side with the blinded soldier, as is now done in the British and French armies. The purpose is that the blind man's family may be informed as to his difficulties, ambitions, training, and needs, so as to prevent the mental deterioration which often results from false sympathy. Friendly relations between the family and teachers will be a potent means of stimulating the interest of the blind man.

A reel of the moving picture about to be shown you will further emphasize the vocational side of the care given our blinded men.

PLAN ADOPTED BY THE GOVERNMENT

The Division of Physical Reconstruction of the Surgeon-General's Office has as its chief Col. Frank Billings, and one of the sections of this division, which has charge of the defects of hearing and speech, is under the directorship of Lieut.-Col. Charles W. Richardson.

After many months of planning, on July 23, 1918, in General Hospital No. 11, Cape May, N. J., a school was established for the defects of hearing and speech in connection with this hospital. The plan of the school is that all defects of hearing and speech

among individual returning soldiers from the A. E. F., as well as those who have become affected in the cantonments, will be instructed or reeducated in this school before they are discharged from the Army. In other words, all soldiers or sailors evidencing deafness or handicaps along the line of this work will, while having their physical disabilities corrected and cured, be reeducated and their handicap raised.

Returned soldiers and sailors having defects of hearing from whatever cause, either from disease or as the result of casualties, are those whose hearing is so reduced that they are handicapped socially and incapacitated industrially. They comprise all the completely deaf and those whose hearing is so reduced that it requires an effort for them to hear and they experience difficulty in communicating through the medium of the spoken voice.

Corrective speech work will be carried out on all neuropathic affections of the voice, all types of paralysis (medical or surgical) and those in that large class of cases due to facial maxillary injuries.

The fundamental principle which has been adopted in connection with this service is to hold the men in the control of the United States Army as pupil-patients until their physical handicap has been relieved. It has been found in studying methods adopted by the various cobelligerents that the retention of the soldier until his handicap is removed, especially for defects of hearing, is the only safe procedure. In the French and Italian Armies, where this method is pursued, all men get their reeducation before they are discharged; therefore, all men with this disqualification or handicap are obliged to accept the type of treatment which is instituted.

In the British Army where men presenting these conditions are discharged, it has been found that reeducation is not taken up by 74 per cent. Indeed, all efforts have been made by the Aural Commission in Great Britain through propaganda, through entertainments, through the offer of meals, etc., to entice these men back to accept reeducation. To the many who wish to work they have even offered evening classes, and yet they do not avail themselves of it, except to the extent of 24 per cent.

We feel, therefore, that centralizing these cases and holding them in the service is by far the safest, sanest and most certain means to accomplish this purpose of reeducation.

It is also well to remember, and we wish it placed strongly before the public, that these men whom we propose to reeducate are not abnormal, but simply have become handicapped

through the loss of a special sense in their service to the common cause of liberty. It should also be understood by the laity that, through the methods which we propose to adopt in reeducating these men, they will not be handicapped; that they will simply be required to read speech with their eyes, instead of hearing speech with their ears. They will be able to take part in all social pleasures, and they will be able to follow almost all the gainful occupations.

In the Section of Defects of Hearing, we will use two principle methods in teaching these people. One is the auricular oral method, which through using the voice and various types of electrical apparatus bring into activity a sense which has been impaired, either through violence, "compression" or neuropsychic conditions.

The second method, and the one that we depend on to the greatest extent, is lip or speech reading.

In those very rare cases in which the individual is too obtuse, or is unable to concentrate, we shall be obliged to make use of the manual method.

Defects of speech arise from various complex causes and comprise those cases which will have to be treated by neuropathic methods, those cases which will have to be met by respiratory methods and those which will have to be taught the principles of phonetics; a large number of cases of face injuries involving the throat will not only have to be treated phonetically but also surgically.

This work of physical and vocational reconstruction was started at Cape May under the immediate medical control of Major John M. Ingersoll, with Capts. Berry and Lampe as assistants. These medical officers, under the Division of Surgery of the Head, will take care of all medical and surgical aspects of pupil-patients.

The educational department has as superintendent Mr. A. C. Manning of Mt. Airy, Miss Enfield Joiner of North Carolina as principal, and four teachers—Miss Mary Louise Wimsatt, Miss Margaret Bodycomb, Miss Clara Louise Rockwell and Miss Mary Thornton.

The first class began on Wednesday, July 24, 1918, with seventeen pupils, many of them completely deaf, most of them extremely hard of hearing. They form a bright, intelligent, enthusiastic group of pupils, ardent in their desire to learn, appreciative of the service being rendered them, and enthusiastic in regard to their prospects for complete rehabilitation.

MONOCULAR RETROBULBAR OPTIC NEURITIS FROM HYPERPLASIA OF THE ETHMOID BONE

REPORT OF THREE CASES WITH OPERATION

DERRICK T. VAIL, M.D.
CINCINNATI

Hyperplasia is defined as an increase of the fibrous connective tissue elements of living anatomic structures due to a low grade of localized inflammation. Delafield and Prudden¹ state, "It is associated with long continued hyperemia or chronic congestion of the organ involved." Hyperplasia is a nonsuppurative process. Skillern,² before the American Laryngological, Rhinological and Otolological Society, has demonstrated by means of many beautiful slides the microscopic difference between hyperplastic and suppurative ethmoiditis. Beck³ has also shown slides prepared from hyperplasia of the ethmoid bone. Welsh⁴ noticed that "the connective tissue stroma of an ethmoid specimen was thickened by fibrous tissue production and acute inflammatory products and there was thrombosis of many small veins." Shambaugh⁵ recognizes the chronic ethmoiditis, which is "primarily a nonsuppurative disease, but with impaired drainage of the cells due to hypertrophy of the mucous membrane of the cell walls." He calls the process hypertrophy, but hyperplasia seems to be the better term. Hypertrophy is defined⁶ as "a simple increase of the size of the elementary structures of a part usually associated with increased function of that part while hyperplasia is an increase of the number of the elementary parts." Hyperplasia of the ethmoid bone is a rarefying osteitis associated with inflammatory swelling and fibrous thickening of the mucous membrane lining its cavities, causing edematous tissue to fill them without the formation of pus. Its limiting wall, called the capsule, is also thickened and the entire organ is enlarged so that the orbital wall is involved as well as the nasal.

1. Delafield and Prudden: Pathology, New York, William Wood & Co., p. 121.

2. Skillern: Tr. Am. Laryngol., Rhinol. and Otol. Soc., 1910, p. 198.

3. Beck: Tr. Am. Laryngol., Rhinol. and Otol. Soc., 1913.

4. Welsh: Tr. Am. Laryngol., Rhinol. and Otol. Soc., 1917 (L. A. Coffin's case).

5. Shambaugh: Tr. Am. Laryngol., Rhinol. and Otol. Soc., 1914, pp. 228-229.

6. Delafield and Prudden: Pathology, p. 93.

Owing to the fact that the middle turbinated bone is attached to the body of the ethmoid by a very narrow strip, this hyperplasia may be and usually is confined largely or entirely to the structure of the ethmoid proper and rarely extends across the isthmus of bone to involve the middle turbinate itself. Because of this fact, it is usually impossible to diagnose hyperplasia of the ethmoid by simple inspection.

Hyperplasia of the ethmoid bone is of itself a comparatively trivial disease. The nasal symptoms are not particularly distressing, for in spite of the sneezing followed by the watery discharge, the transitory impairment of nasal respiration, and the paroxysmal reflex cough, the patient is not greatly annoyed. He may complain of being sensitive to drafts and subject to head colds and also of being kept awake at nights by having one side of his nose stopped up, a condition requiring him to turn over on the other side. There is no pain, no purulent discharge and no impairment of the sense of smell.

Owing to the location and structure of the ethmoid bone, and the truly remarkable variation in the size and arrangement of its pneumatic spaces, and also to the fact that the optic foramen lies in many instances in juxtaposition to its large posterior cell (indeed, may traverse it), hyperplastic inflammation may here assume an important rôle and produce blindness by causing retrobulbar optic neuritis.

The object of this paper is to establish clearly the relation between hyperplasia of the ethmoid bone and acute retrobulbar optic neuritis and to point out the importance of early diagnosis and early operation.

SYMPTOMS

The ocular symptoms of inflammation of that part of the optic nerve which traverses the optic foramen (or what is better termed "the optic canal," for it averages 15 mm. in length) are definite and easily recognized. The patient complains of foggy vision affecting one eye, which rather rapidly becomes worse, so that in a few days he may be entirely blind in the eye. There is usually slight pain on directing his eye to the limit of motion and a slight pain or uneasiness referred to the temple on that side and back of the eyeball. The one symptom which impels him to seek the ophthalmologist is the fear of blindness and the very positive and annoying loss of vision affecting one eye. There are no nasal symptoms, hence the rhinologist will not be sought.

In the fulminating type of the disease there appears sudden severe pain involving all the branches of the fifth nerve, particularly the filaments of the first division, and with it occurs blurred vision, which in a few hours progresses to total blindness of the affected eye.

The objective findings which establish the diagnosis in the acute stage are of two kinds, positive and negative. The positive findings are: (a) monocular blindness; (b) sluggish response of the pupil to direct test, and (c) dull pain on deep pressure.

(a) Monocular blindness: When there is vision present, it is always eccentric. The periphery may still be normal with a very large total scotoma occupying the central area, as shown by the accompanying field charts of my cases. The scotoma is probably relative before it becomes positive, so that if the case

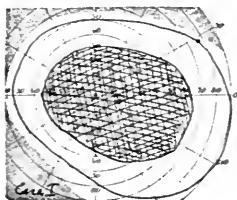


Figure 1.

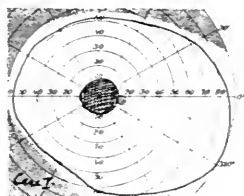


Figure 2.

Fig. 1 (Case 1).—Field of vision as recorded five days before operation.

Fig. 2 (Case 1).—Field of vision as recorded fifteen days after operation.

appears early enough, we should find a partial scotoma for colors in the center with enlargement of the blind spot. When the patient comes later, as is usually the case, the blind spot is included in the scotoma.

(b) Sluggishly reacting pupil in the affected eye: This slow reaction of the pupil is present to the direct test and not to the indirect or associated tests. The pupils are usually of equal size because the pathway of the third nerve down from the sphincter nucleus in the brain to the iris is not involved and the light impulse in the unaffected eye causes an equal efferent response in both pupils. The pupillomotor fibers of the optic nerve in the affected eye are inhibited in function by the disease present in the optic nerve at the apex of the orbit and hence tardily convey the light impulse upward to the brain, or not at all, as the case may be.

(c) The symptom of slight tenderness referred to the apex of the orbit when making pressure against the eyeball through the closed eyelids is not always present.

The negative findings are quite important, for they assist greatly in establishing a diagnosis. They must be studied in relation to the positive. They are: (1) normal disk and fundus, and (2) normal middle turbinate.

1. Normal appearing optic disk and fundus in the eye affected with central scotoma or blindness is characteristic of this disease. Later on, usually after a month following the onset of the trouble, there will appear beginning atrophy of the optic disk. This atrophy progresses in proportion to the severity of the retrobulbar neuritis that preceded it and may be complete. Usually it is partial. A careful study of its extent will reveal

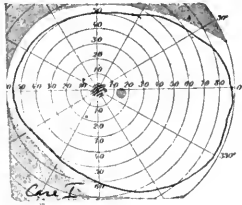


Figure 3.

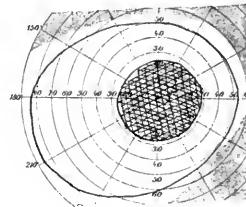


Figure 4.

Fig. 3 (Case 1).—Field of vision forty-three days after operation.
Fig. 4 (Case 2).—Field of vision as recorded one day before operation.

that it involves the temple side of the disk representing a wedge or a sector of from one-quarter to two-thirds of that side of the disk and is a white atrophy with clearly cut disk margin, in other words, a typical primary descending atrophy. A small sector of the disk on the nasal side may still retain a degree of pinkness.

2. In the case of a normal appearing middle turbinate body with no discharge present in the nose, the turbinate is usually not visibly enlarged, nor is it discolored. It is not usually impacted against the septum and there appears ample breathing space and normal nasal function. If, however, the body of the middle turbinate be shrunk with cocaine and epinephrin, one may find the evidence of hyperplasia of the ethmoid proper by observing small polypi hidden under it.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes toxic amblyopia, congenital amblyopia, malingering, hysterical blindness of one eye, monocular optic neuritis from systemic infections and monocular blindness from fracture at the base.

In toxic amblyopia the history of inordinate use of alcohol and tobacco is usually easily determined or the taking of wood alcohol, large doses of quinin, etc., is admitted. Toxic amblyopia is much slower in its course than amblyopia due to ethmoid disease. Moreover, it is almost invariably expressed in both eyes, although with different degrees of severity.

In congenital amblyopia affecting one eye, the differential diagnosis is easily made if the patient has been aware of affected vision in one eye for a long period of time. Occasionally such patients discover the amblyopia by accident only a short time before consulting the ophthalmologist, or the latter may discover

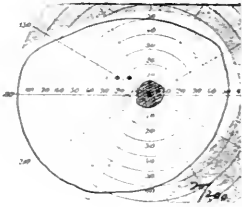


Figure 5.

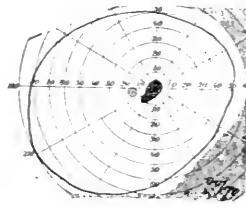


Figure 6.

Fig. 5 (Case 2).—Field of vision thirty-seven days after operation.

Fig. 6 (Case 2).—Field of vision fourteen months after operation.

it for the first time during the process of examination of the patient's eyesight. The cases of congenital amblyopia will show no central scotoma even by use of the 3 mm. disk. His fields of vision for white and colors will be normal in periphery and center. Congenital amblyopia is stationary and presents no sign of atrophy at any time, while the disease under discussion is never stationary and the disk eventually shows distinct atrophy.

Malingering blindness of one eye will be discovered by an astute ophthalmologist who knows how to conduct the proper tests and uses them.

Hysterical blindness in one eye is either partial or complete. If partial, the field of vision is usually typically contracted with no evidence of central scotoma. If total the tests applied for detection of malingering blindness will suffice.

Monocular optic neuritis from systemic infection or disease is diagnosed by the absence of central scotoma and the history of such infection, for example, measles, typhoid fever, scarlet fever, etc. In some cases, the optic neuritis and neuroretinitis are discovered to be due to albuminuria or diabetes which may produce the same picture as infections. The optic neuritis due to local infections from showers of germs thrown into the circulation from a suppurative focus in the body as from diseased tonsils, diseased bone as at the apexes of the teeth, etc., is differentiated also by the absence of central scotoma or rapidly progressing blindness and by the presence of a frank neuroretinitis. It is sometimes difficult to locate the origin of these infections. In one of the cases reported in this paper, the patient had an acute empyema of the ethmoid cells which produced no nasal symptoms worthy of note and which was discovered at the time of the operation. An able roentgenologist must be engaged to make careful plates and submit them along with his interpretation.

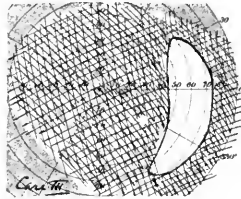


Fig. 7 (Case 3).—Field of vision as recorded seven days after operation.

Other types of optic neuritis such as occur in brain tumor with increased cerebral pressure or from pituitary disease or meningitis, etc., need not be dwelt on here.

The type of optic nerve lesion which most nearly simulates that due to hyperplasia of the ethmoid is the traumatic in which there is a fracture of the base involving the bony structure at or near the apex of the orbit. I have recently seen a case in which the roentgen ray showed line of fracture involving the roof of the orbit running back to the bony apex. The fracture was due to a fall into an excavated pit, the patient striking the right temple on a block of cement. There was total blindness of the right eye. The normal optic disk and retina were observed ten days after the accident. Signs of atrophy appeared on the twenty-first day and total atrophy on the twenty-eighth day, the eye remaining blind. In this case, there was not the slightest sign of optic neuritis as seen with the ophthalmoscope. The

chiasm, so that the compression of the nerve in its foramen does not affect the chiasm, and the clinical manifestations are monocular.

The fibers which supply the macula lutea of the retina lie in the center of the optic nerve at the foramen, but as the eyeball is approached from behind, they are seen to shift gradually toward the temple side, so that at the disk as seen with the ophthalmoscope they occupy the wedgeshaped temple fourth or third, and the artery and vein are at the center.

OPHTHALMOSCOPIC EXAMINATION

Why is it that the ophthalmoscopic examination is negative in the active stage of this disease? The answer is that the central artery of the retina and its vein exist in the nerve only for a distance of 15 mm. behind the eyeball. Beyond that, they lie outside the sheath of the nerve. The transverse optic neuritis and compression exist nearly an inch further back and the retinal circulation is not impeded by it. Within thirty days after the disease announces itself, atrophy of the disk is seen and it appears as a primary atrophy.

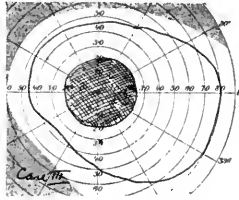


Fig. 9 (Case 3).—Field of vision thirteen months after operation.

SIGNS OF THE DISEASE

Why is central scotoma an early sign of the disease?

The papillomacular fibers of the optic nerve are the finest, most highly specialized and most vulnerable. Swelling of the bony wall of the optic foramen causes as much pressure in the center of the nerve contained therein as at the periphery, according to the well known law of physics. The first to suffer are these delicate fibers supplying the macular region of the retina. It is not inconsistent with the theory of compression to have interstitial neuritis as described by Collins and Mayou, intra-neural exudation, perineuritis, varicosity of the axis cylinders, fatty degeneration of the nerve cells, capillary thrombosis, etc. But I take it that these phenomena are secondary to the strangu-

lation and are natural in pathologic sequence. They follow the compression and in turn are followed by the atrophy. The point I wish to emphasize is that infection of the nerve is not necessary to produce optic neuritis within the optic canal. Mechanical pressure is sufficient. It is undeniable, however, that in certain cases the optic nerve may share in the localized inflammation because of mere identity of its bony collar with the ethmoid wall.

Arnold Knapp⁷ recognizes the types of cases reported in this paper, stating that there is retrobulbar optic neuritis in which central scotoma is the characteristic functional defect. He further states:⁸

Optic neuritis of nasal origin occurs in two forms; one during the course of suppurative sinusitis, the other where there is a typical retrobulbar neuritis and the rhinoscopic examination is negative. In the latter cases, exploratory operation reveals a latent infection. The more acute the case the more rapid the loss of vision and the better the prognosis. The effect of the nasal operation is remarkable. The nasal diagnosis in these acute cases is often difficult; even with negative rhinoscopic findings an exploratory operation must frequently be undertaken.

Since my paper on "Optic Neuritis from Intranasal Disease" was read before this Society in 1901,⁹ I have seen many cases illustrating the relation between ethmoiditis and acute retrobulbar neuritis, but I shall report three typical cases that came under my observation within three weeks of one another. All three patients were operated on at once, in spite of the fact that in each of them the ethmoid appeared normal as viewed rhinoscopically.

REPORT OF CASES

CASE 1.—L. W. E., male, aged 35, a telegraph operator, was seen March 14, 1917.

History: Twelve days previous to consultation while working in his office, he had noticed foggy vision in his right eye. No pain was felt at any time. There was no history of trauma. The patient had noticed deterioration of vision from day to day. He was a total abstainer from alcohol, tobacco and coffee. He denied syphilis. There were no nasal symptoms.

Examination of Eyes: External inspection was negative; tension was normal in the right and left eyes. The pupils measured 3 mm. in the right and left eyes. The right pupil responded feebly to direct test and readily to indirect. The left pupil reacted quickly to direct tests and not at all to indirect.

7. Knapp, Arnold: *Medical Ophthalmology*, p. 379.

8. Knapp, Arnold: *Medical Ophthalmology*, p. 382.

9. Vail, D. T.: *Am. Jour. Ophth. (Alt)*, 1901.

The ocular motility was unaffected. Vision: The right eye could not count fingers. In the left eye, the vision was 20/20 corrected and read Ja. No. 1. As for the field of vision in the right eye, the periphery was nearly normal, and there was a large central scotoma extending approximately 40 degrees in all directions from the center (Figure 1).

Examination of Eyes Under Homatropin: The pupils dilated equally and ad maximum, and the dioptric media were clear. The fundus in each eye appeared entirely normal in every regard and no difference was noted by comparing the optic disks.

Nose: The inspection was negative. The turbinated bodies appeared unaffected, and the breathing space was normal. No discharge was present, and no difference was noted between the two nasal chambers.

Report of Roentgenologist (Dr. Lange): The frontals and antrums were clear. The oblique exposure of the ethmoid showed distinct clouding of the posterior ethmoid cells on the right side. The sella turcica was normal.

Diagnosis: Monocular blindness from nonsuppurative ethmoidal disease on the right side.

Operation: March 19, 1917, the right ethmoidal labyrinth was exenterated by Mosher's technic. Twenty-four hours after operation, the patient stated that his vision was improved.

April 3, the vision was 20/50 and the central scotoma did not extend more than 10 degrees from the fixation point (Figure 2).

April 17, the vision was 20/30.

May 1, the vision was 20/30. The central scotoma was relative and there were signs of slight atrophy on the temple side of the disk (Figure 3).

May 29, 1918, one year later, the vision was 20/20 and read Ja. No. 1. The ophthalmoscope revealed distinct pallor of the disk, especially involving the temple side. The right disk was nearly white, the left was quite rosy. In spite of the atrophy, the vision was 20/20 and Ja. No. 1, and the fields of vision were entirely normal for white and colors. There was never any discharge from the nose after operation, and the field of operation was clean and normal appearing.

CASE 2.—Miss V. M., aged 18, a schoolteacher, was seen March 20, 1917.

History: Two weeks previous to consultation, she had had a mild attack of "pink-eye" and "cold in the head." There had been some pain in the left eyeball whenever she directed the eyes to the extreme field of fixation. This pain still persisted. Five days before, the vision of the left eye had become dim.

Examination of Eyes: The right eye appeared normal, the pupil being $2\frac{1}{2}$ mm. and responded to direct test. The vision in this eye was 20/20 and Ja. No. 1. The field of vision for white and colors was normal in the right eye. The ophthalmoscopic examination revealed a normal disk and fundus.

Left Eye: There was orbital tenderness on deep pressure against the eyeball. The pupil measured 2.5 mm. and was slug-

gish in reaction. The tension was normal. Ophthalmoscopic examination revealed a distinctly swollen optic disk. The veins were swollen and tortuous. The details of the fundus were quite hazy because of the presence of neuroretinitis. The vision was barely 20/200. The field of vision at the periphery was normal, but there was a large absolute central scotoma present (Figure 4).

Nose: There was ample breathing space. The turbinated bodies appeared normal and alike on the two sides. No secretion was present.

Report of Roentgenologist (Dr. Lange): The frontals and antrums were clear. The left ethmoid taken by oblique exposure showed a distinct cloudiness. The right appeared normal.

Diagnosis: Left optic neuritis from ethmoid disease.

Operation: March 21, a complete exenteration of the left ethmoid and sphenoid was done under cocain anesthesia. The cells contained pus.

March 24, the left optic disk appeared less swollen.

April 7, the patient could barely see at 20/200. There were signs of beginning atrophy of the disk.

April 27, the left optic neuritis had subsided and atrophy was becoming established. The patient still had absolute central scotoma, though not so large as at first noted (Figure 5). The vision was 20/200.

June 22, partial atrophy of the optic nerve was especially noted on the temple side of the disk. The vision was 20/100. The nose appeared free from discharge. (The day following the nasal operation, there was quite a flow of creamy pus from the sphenoid and ethmoid region.)

May 18, 1918, fourteen months later, the right optic disk was normal and vision was 20/20 and Ja. No. 1. The left disk was uniformly pale. The vision was 20/70, with a small central scotoma (Figure 6). The nose was free from discharge and appeared healthy.

Comment: This was undoubtedly a case of optic neuritis from ethmoid disease, as there was a suppurative process at work in the postethmoidal and sphenoidal cells. The rhinoscopic examination before operation did not disclose the suppuration. It was revealed by the operation.

CASE 3.—Miss M. S., aged 60, with no occupation, was seen May 4, 1917.

History: Without previous warning and from unknown cause, the right eye suddenly had become blind three weeks previous to consultation. The only other sensation was that of a dull pain in the right temple and brow. The blindness of the right eye was complete, as there was no light perception.

Examination of Eyes: The external inspection was negative. The pupils were nearly equal in size, the right measuring 4 mm. and the left 3.5 mm. The right did not respond to light, but it did respond consensually and associately. The eyes appeared straight, and motility was normal.

The ophthalmoscopic examination was entirely negative in the right and left eye. The vision in the right was nil. The left was 20/20 corrected. The tension was normal in each eye, and the field of vision in the left eye was normal for white and colors.

Nose: The nasal chambers appeared entirely normal. There was no history of chronic nasal disease.

Diagnosis: Monocular blindness from concealed ethmoid disease.

Operation: May 4, 1917, exenteration of ethmoidal labyrinth was done, and the sphenoidal cell on the right side was opened.

May 11, there was a slight response of the pupil to light. Hand movements were detected in the extreme temple field (Figure 7).

May 18, the patient could count fingers eccentrically. She still had central scotoma (Figure 8).

June 15, 1918, thirteen months later, the optic disk was found atrophied on the temple side. There was still an absolute central scotoma present as shown on the accompanying chart (Figure 9), but the peripheral field was restored. The pupils were normal in size and the right readily responded to light. The nose appeared normal, and no purulent discharge was present at any time before or after the operation.

Vision: The patient could count fingers at 10 feet by eccentric fixation.

CONCLUSION

The cases reported illustrate two types of monocular optic neuritis from ethmoidal disease. In the one type illustrated in Case 2, there was a frank exudative neuroretinitis associated with functional blindness in the center of the visual field, due to a suppurative process at work in the posterior accessory sinuses of the nose that lie in proximity to the optic nerve. The exudation was doubtless due to bacterial influences and the central scotoma to the swelling of the nerve (interstitial neuritis) within the bony optic canal.

In the other type illustrated in Cases 1 and 3, there was no bacterial invasion of the nerve present, but there was swelling of the orbital wall of the posterior ethmoidal or sphenoidal cell, which pressed the optic nerve in its passage through the optic foramen and caused a strangulation of the nerve at this point. This compression no doubt produces a transverse optic neuritis which, while not due to bacterial invasion, is nevertheless destructive to vision and is followed by more or less permanent atrophy of the optic nerve. The nasal disease that produces this phenomenon is a nonsuppurative one, namely, hyperplasia of the ethmoid bone.

The disease should be recognized and operation on the ethmoid performed at once in spite of its being normal in appearance. The diagnosis is made solely from ocular findings.

The plea I would finally make is: Operate at once. The prognosis is good as to restoration of vision if the operation is performed during the acute stage, but if it is delayed until atrophy sets in, the prognosis is bad.¹⁰

DISCUSSION

DR. JOSEPH S. LICHTENBERG, Kansas City, Mo.: As usual, a paper from Dr. Vail has covered the ground so thoroughly there is nothing to be discussed. Unfortunately, Dr. Vail did not read all the paper. I looked over it and you will find much more there when you read the Transactions. The report is of a very interesting class of patients to which the ordinary ophthalmologist pays little attention.

Considering the cases anatomically, the work of Onodi and of Loeb, shows these cells sometimes lie in close apposition to the optic nerve as mentioned, but the ethmoid and sphenoid cells may be so branched that it is possible the cells from one side may affect the nerve on the other side. You will remember the arrangement of the macular bundle within the optic nerve, how it goes over the outer side as it enters the eyeball, and this accounts for the peculiar symptom of the enlarged blind spot. It also accounts for the symptoms in Case 2. Where the hyperplasia is present we have the cause as in Cases 1 and 3. When we consider these cases from a pathologic standpoint, Jonathan Wright and others have worked this out very nicely.

Coming to the symptomatology of the doctor's cases, they are more of the fulminating type. These are rather rare, the chronic or subacute being more frequent than we acknowledge. In my experience the first thing noticed is a difficulty in vision. These people go from one man to another, have glasses fitted, etc. The next and most important symptom is the enlargement of the blind spot. Wherever you have this it is most probably from the ethmoid or the sphenoid, as in Dr. Vail's paper. Taking the blind spot is not as easy as expected. The campimeter of Peters is good, but cannot be held steadily. That described by Gradle, which fixes the head, is easier. Of course, the central scotoma is the next thing, and the diminished vision is the next.

I will report one case, which happens to be bilateral. A man, aged 32, a blacksmith, comes in saying he looks into the fire too much. Fundus examination and refractive examination negative. In the hospital the Wassermann reaction, blood and everything else are negative. A scotoma developed on the left side. The roentgenogram revealed a distinct shadow in both ethmoid regions. Both cleared up with perfect recovery, vision now 20/20, and the man was back at work after exenteration of the ethmoid cells and drainage of the sphenoid sinuses on both sides (Dr. Alvin Lorie).

10. Since my writing of this paper, Greenfield Sluder's book on "Headaches and Eye Disorders of Nasal Origin" (St. Louis, C. V. Mosby Company, 1918) has been published and I note many strikingly similar arguments to sustain the proposition that hyperplasia of the sphenoidal ethmoid bones causes visual and other disturbances from compression of the trunks of the anterior group of cranial nerves at the foramina of exit. My paper was written without knowledge of Sluder's work in the same field, which accounts for lack of reference to his masterly production. His book covers a much wider range and, being written by a rhinologist of renown, will receive and merit the highest commendation.

DR. JOSEPH C. BECK, Chicago: Coming over from Chicago I had an opportunity to talk this over with Dr. Vail and to compare notes. He told me it struck him as peculiar that in a few years I had just one case which is published in the *Annals of Ophthalmology*. It was very much like Dr. Lichtenberg's case, very acute. Within a few weeks I saw a case, the history given to me by a good ophthalmologist. He could see nothing in the eyes and nothing in the nose. She was sent to me, and with Dr. E. V. L. Brown we decided to see what we could find. The roentgenogram, spinal puncture, etc., were negative. At the advice of Dr. Brown I did the exenteration on both sides with remarkable changes. She could see nothing, and five hours afterward, when Dr. Pollock entered the room, she said: "I see you." He reported to me 20/30 and 20/40 vision. It is the one case, and I do see a good many eye cases. It must be the anatomic condition. Another point is that the roentgen-ray examination is negative. Taking Linne's oblique method, the shadow is made. I did find a slight shadow in the lining membrane, and you must remember that it is not osteitis, but the opposite. The ethmoid should have shown a rarefying process.

DR. FRANK R. SPENCER, Boulder, Colo.: I would like to emphasize that it has not been my experience that I have difficulty in finding the pathology in the nose. In 10 per cent. of the cases I have found a normal nose, but in 90 per cent. have had no difficulty in finding nasal pathology. We have evidence of a hyperplastic ethmoiditis.

DR. W. W. PEARSON, Des Moines, Ia.: It is with hesitation I report a case of this type. A thorough examination had been made, roentgen ray and everything, and no clinical signs of anything wrong. Examination of the nose revealed a spur along the left side buried in the middle turbinate. Vision 20/100 in one eye and standard in the other. Finding everything negative, duration of the impairment of the vision being about ten days, and in the hope of being of some service I recommended and performed a resection of the septum. The man had none of the stigmata of hysteria, and in the course of the removal of the spur the statement was made by the patient, "My vision is better in that eye." It did not impress me at the time. The next day he was very much better and in the course of a week the vision was wholly restored. I reported this to Dr. Gifford, who had made a similar observation which he was unable to explain; he thought possibly there might be some gas in the occluded ethmoid cell to account for it. I hesitated to report this case following Colonel Wood's case report, fearing some one might think I am an osteopath.

DR. THOMAS J. GALLAHER, Denver: Dr. Vail's paper is indeed timely, and recognition of this condition is most important to the patient. I have had three such cases during the last year. The rhinologist must constantly keep in mind that there may be irregularities in the anatomy of the sinuses. In some cases the sphenoid sinus is in closer relation to the opposite optic nerve than it is to the optic nerve of the same side. It is therefore necessary that both nostrils be critically examined to ascertain if the cause is due to nasal condition, involving one or both eyes. I have seen one case in which the eye was involved secondary to sphenoidal disease in which the external rectus was also paralyzed. There must be no delay in surgical interference. It may be necessary to operate on the ethmoids and sphenoid of one side and in addition the sphenoid on the opposite side even if the trouble is limited to one eye.

DR. VAIL (closing discussion): I have nothing to add excepting to thank the gentleman who opened the discussion and those who followed.

HYOSCIN-MORPHIN ANALGESIA FOR OPHTHALMIC OPERATIONS

HARRY VANDERBILT WÜRDEMANN, M.D., CAPT., M. C., U. S. A.

SEATTLE

"He sleeps well who knows not that he sleeps ill."—Maxim.

Persons who have been suffering from glaucoma with its disturbing symptoms of pain and obscuration of vision and those in whom the slowly progressive loss of sight from cataract occurs, generally become nervous and some of them indeed demand the utmost mental skill of the surgeon for their psychic care as well as his dexterity and medical knowledge. This general nervousness is certainly most pronounced on the day of the operation and is perhaps enhanced somewhat by the necessary preliminary treatment in securing an aseptic field for the operation.

AUTHOR'S OBSERVATIONS

I have had numerous and grievous experiences. Looking into the past, I remember quite a number of cases of postoperative mania; in several of them, leading to suicide.

A most beneficial change has occurred in my private practice during something over the last two years, since when, almost as a routine, I have been using hyoscin and morphin hypodermically before the operation. This short paper takes up nothing but the personal experiences of the writer with a brief series of cases which deal with two of his clinic days, as for want of time he has not been able to look up a full list of his case histories. He contrasts these two days' experiences, and also gives several other incidental cases for this purpose.

TECHNIC OF ADMINISTRATION

After the usual preparation of forty-eight or twenty-four hours has been completed, one hour before the time set for the operation $\frac{1}{4}$ grain of morphin, and $\frac{1}{100}$ grain of hyoscin are given hypodermically. By this time the patient is somnolent but most of them are able to make volitional movements of the eyes and to answer questions. Some of them become comatose. For local anesthesia at the time of operation, three instillations of 5 per cent. cocain muriate solution are made, usually with 1 drop

of 1:1,000 adrenalin solution, and the operation is commenced. Most of these patients know at the time that the work is being done, but quite a number are absolutely asleep and do not know anything about the operation. The somnolent condition usually lasts about six hours. Sometimes it is prolonged for twenty-four hours. The patients are sufficiently under the influence of the hypnotic so that they do not talk and they are absolutely quiet. Even in those cases in which a comatose condition has been produced, no difficulty has been encountered during the operative procedure. It is found that the eyes are not rotated strongly upward as they are during ether narcosis and that the fixation forceps are quite sufficient to steady the eye during the corneal incision, the iridectomy or the trephining.

UNTOWARD EFFECTS UNCOMMON

In only one instance has any untoward effect been observed and that was vomiting before the proposed operation which perhaps, in this instance, may not be ascribed to the morphin but rather to an indigestion, this patient having been rather hurriedly prepared for the operative clinic. In this case the operation was passed over to the next day and at that time no effect other than that of narcosis was observed.

I have been agreeably surprised to find that there has been no nausea or vomiting in any cases following the operation. I have likewise not noted any prolonged effect on the pupil by the morphin, the iris always responding to the mydriatic effect of the cocain.

Many of my operations have been watched by visiting surgeons, particularly, the cataract clinics which I have held once a year, when I have collected a number of cases for this purpose.

We will now take the two clinic days for contrast, the first in 1914 and the other one in 1918.

Nov. 17, 1914, I asked a number of my confrères to observe my methods in cataract operations and four cases were brought together for a clinic at the Swedish Hospital.

REPORT OF CASES

CASE 1.—H. H. This operation was a discission which proceeded normally, the patient not being nervous.

CASE 2.—Mrs. A. S. MacD. This operation was an extraction in the capsule and the procedure was fairly normal, the patient being no more nervous than usual; but within a few hours afterward she squeezed her eyelids and caused an iris prolapse.

CASE 3.—Mrs. D. G. The patient was extremely nervous. I attempted an extraction, but the lens would not come out. A large bead of vitreous extruded and I put in two sutures through the conjunctival flap, closed the eye, doing the extraction on November 30 of the same year. In August, 1917, I extracted the cataract in the other eye, at that time using hyoscin and morphin. The patient was practically comatose. The lens came out without undue pressure, immediately following the iridectomy; in fact, it presented at the time and was simply lifted out in its capsule. She received two doses, namely, $\frac{1}{2}$ grain of morphin with $\frac{1}{50}$ grain of hyoscin, and she slept for two days afterward. I do not consider now that this large dose is necessary.

CASE 4.—Mr. J. D. H., was an extremely nervous man. I had, in this case, the misfortune before an appreciative audience to have self expression of the lens performed by the patient himself, immediately following the corneal incision. Healing occurred and he obtained 6/60 vision with his correction, but two years later detachment of the retina was found. I operated on the other eye March 1, 1918, using hyoscin and morphin and the local anesthetic. The patient was quiet and I performed a complicated extraction which had to be done by the lens loop as the vitreous was fluid. With this eye, too, the patient obtained only a mediocre result, namely, 6/60 vision with correction, but he is able to get about the streets and to do his home work.

In contrast to the foregoing cases I will now take up a clinic of seven cases which I held at the Minor Hospital, Jan. 11, 1918, in which the conditions were quite as complicated but in which there is a most beneficial contrast.

CASE 5.—Mrs. H. E. H. This was an extraction with discission of the capsule. The patient was absolutely calm and the subsequent healing and result were excellent.

CASE 6.—Mrs. H. M. MacD. The patient was an extremely nervous woman. The condition required an extraction in the capsule and corneal incision. There was a small bead of vitreous, but no loss. The patient was unconscious during the time of the operation. Good end-result followed.

CASE 7.—Mrs. D. E. W. The condition was one of high myopia; expression in the capsule, with slight loss of fluid vitreous. One stitch was taken with good immediate result, but several months later, optic nerve atrophy followed. This patient was practically likewise unconscious but could answer questions and direct eyes.

CASE 8.—J. T. B. There was normal perfect expression in the capsule. The patient was fully conscious, but did not feel anything. Good results followed.

CASE 9.—Mrs. C. M. S. Another extremely nervous woman, normal expression in the capsule, but on the fifth day afterward prolapse of the iris occurred and very slow healing of the wound followed. This resulted from the patient squeezing the eye at that date. Good end-results occurred in this case.

CASE 10.—Mrs. C. D. In this case there was nausea when the patient was brought into the operating room and therefore she was returned to her room. The operation was performed two days later. During the operation there was self expulsion of the lens and loss of vitreous. We did not give her any hypodermic injection on this occasion on account of the previous day's experience, but would certainly have done better if we had, for the end-result was practically nil as regards sight.

CASE 11.—E. G. W. In this case, I performed a double Elliot operation for glaucoma. The patient was conscious but very drowsy and we had no trouble.

In the foregoing I have given but few details regarding the operation or result, but have simply referred to the effect of the drug in aiding the operation and in quieting the patient.

OTHER CASES FOR COMPARISON

I would also like to contrast the manner and experience of a patient, Mrs. E. B. MacD., who had glaucoma. At the first operation, June 17, 1918, trephining into both eyes was done while the patient was under the influence of the drugs, and in the second, July 5, 1918, when she returned with the lens of the left eye dislocated into the anterior chamber at which time it was extracted; through an error in direction the drug was not administered. At the first operation the patient was tractable and gave no trouble; and during the second she was extremely nervous, giving us great difficulty throughout the operation and during the following twenty-four hours.

Another case yet under my care is cited on account of the profound narcosis produced and the care at the time of the operation contrasted with the great amount of trouble before and after. This case was that of a Jewess, Mrs. H. K., aged 70, of South Carolina. The examination was made May 13, 1918. The patient had diabetes and cataract of both eyes; she was referred to Dr. Phinzy F. Calhoun of Atlanta, for operation. He sent her back to me, and she came on July 11. The amount of sugar was found to be slight. The patient was placed under the care of Dr. William C. Heussy. The percentage of sugar was reduced to nothing by diet and treatment. Operation: Extraction in the capsule July 16, at the Minor Private Hospital was performed under profound narcosis by $\frac{1}{4}$ grain of morphin,

and $\frac{1}{100}$ grain of hyoscin. The operating room experience was absolutely normal and the result was first class. Before the operation, she would ring me up by telephone four or five times a day. Had all her friends in flocks around. Had the Rabbi "up in the air," also became mixed up with the Catholic Sisterhood and had the Mother Superior in the same condition. The two days in the hospital in preparation were ones of constant ferment, almost causing explosions of temper and loss of patience in every one of the nurses and all of those connected with the case. It has been much the same since, despite the fact that she was kept under the influence of morphin and bromids for ten days; three trained nurses were in attendance. July 31 the patient left the hospital in good condition for a hotel with a trained nurse. Certainly this case could not have been operated on by local anesthesia only and you all know the difficulty of cataract extraction under ether narcosis.

COMMENT

Thus my experience with the administration of these two drugs before the operation has seemingly been most favorable, and it is recommended to those who are not using the procedure, all of which is likewise a contrast to the words of Othello:

"Not poppy, nor mandragora nor all the drowsy syrups of the world shall ever medicine thee to that sweet sleep which thou owedst yesterday!"

DISCUSSION

DR. O. S. FOWLER, Denver: I do not know whether I, as a general surgeon, can say anything of value. I know nothing of your operations for cataract and glaucoma; but Dr. Jackson invited me to say what I might to you. One thing Dr Würdemann spoke of is that the patients are often exceedingly nervous over the operation. They have heard of unfortunate results and sometimes hear discouraging statements from the profession. I recall a case of hernia in which the patient had been told fifteen years before it would kill him. There was no reason why it should kill him, and yet he had this dread all this time. The advantages of quieting the patient are, to my mind, obvious. He goes through it with calmness and it enhances the chances of success. It lessens the shock of the operation. I believe you do not have to consider the amount of shock, but in general surgery it makes a great difference. I might mention, to demonstrate the small amount of shock, some years ago in a period of three weeks I had several cases of gallbladder operations, which consisted of removing the stones and drainage, and eight patients required gas enemas following the operations. Three patients required none at all. Surgeons will tell you of the intense shock that follows operations with local anesthesia at times. In a man in whom I amputated both legs, his pulse was 84 at the beginning and 82 at the end. Most surgeons give an opiate of $\frac{1}{4}$ grain of morphin following major operations. I believe the same patient giving nothing before and nothing after the operation is less nervous after than before the operation. At this time they have gone through the storm and they are quieter, even with the additional pain of the operation than

in contemplation of it. It minimizes the danger of your operation. As Dr. Würdemann mentioned, the patient is capable of conversing during the operation. They understand when I tell them to bear down in hernia operations, to determination if there is a second sac, etc. I have used it in all ages, in children from 2 to 10 years, and in old people up to 89. They say if they had it to do again they would prefer a local anesthesia. In one instance I have operated on seven hernias in one family because the first was successful.

The dosage depends on the body weight of the patient as well as the mental and nervous make up and the operation proposed. I give a different one from the essayist. Instead of giving the No. 1, which is $\frac{1}{4}$ grain of morphin with $\frac{1}{100}$ grain of hyoscin. I advise the giving of a No. 2 forty-five minutes before taking the patient to the operating room. If there are no untoward effects, I give another No. 2, or $\frac{1}{8}$ grain of morphin, just before going to the operating room. In very difficult operations I usually give another $\frac{1}{8}$ grain after the operation is under way. That makes $\frac{3}{8}$ of a grain of morphin they receive. It lessens the shock and the patients usually sleep several hours, although they can be roused when you care to ask them anything. I believe the hyoscin clouds the memory. I have asked them questions and they have answered intelligently and next day would not remember the operation and will ask whether or not it has been done. All this is carried out by the experience of men who have combined local anesthesia with an analgesic. In 400 major operations of hernias or larger operations, and 500 minor operations in which we have given only one dose of morphin we have had no untoward effects. In two cases there was an error by the nurse. A No. 2 is one-half that of a No. 1. In two cases, a nurse who had been told to give a No. 2 gave a No. 1 twice, with the idea that two $\frac{1}{4}$'s made $\frac{1}{2}$ instead of $\frac{1}{2}$ grain. In one case the hyoscin made the man so we had to hold him on the table, but no bad effects followed.

DR. LUTHER C. PETER, Philadelphia: For some years past, in my association with the late Dr. Wendell Reber, we endeavored to find a suitable form of narco-anesthesia for eye cases. The formula which Dr. Würdemann has suggested was the first method used with the results which he has given us today. However, as in his experience, we occasionally found a patient who would come into the operating room in a delirious condition, and occasionally the narcosis lasted for a considerable period after the operation. The method which we evolved, and which I have now been using for a number of years is as follows:

Each patient receives two hours before operation 10 grains of chloral hydrate and 15 grains of sodium bromid. One hour before operation the same dose is administered. One-half hour before operation women patients receive $\frac{1}{6}$ grain of morphin and $\frac{1}{150}$ grain of atropin sulphate hypodermically; male patients receive $\frac{1}{4}$ grain of morphin and $\frac{1}{150}$ grain of atropin. This preparation brings the patient into the operating room in a perfectly quiet and more or less somnolent state. As a rule, they are entirely free from worry; are able to respond to the surgeon's questions and readily cooperate, which is an essential in ophthalmologic surgery. By this method I feel that we have accomplished two things: (1) the "squeezer" is practically eliminated, and (2) the patient is free from apprehension and the operation is accomplished without many of the unfortunate incidents which occur in ophthalmologic surgery.

DR. WÜRDEMANN (closing discussion): I think that all the points have been covered. I would like to suggest this procedure in intranasal operations, as resection of the septum and in tonsillectomy. In either case $\frac{1}{4}$ grain of morphin and $\frac{1}{100}$ grain of hyposcin is sufficient to quiet the patient.

THE USE OF TUBERCULIN IN OCULAR TUBERCULOSIS

A RÉSUMÉ OF TEN YEARS' EXPERIENCE IN ITS USE

LUTHER C. PETER, M.D.
PHILADELPHIA

Some years ago, after reading a paper on "The Use of Tuberculin in Ophthalmic Practice," one of the gentlemen who discussed the paper, said he rarely saw a tubercular eye condition in his practice. It is quite possible that any given oculist may but infrequently be called on to treat tuberculosis of the eye, but one can safely say today that the average man sees a fairly large number of patients whose ocular conditions are of tubercular origin. Our increased knowledge of heretofore obscure and unclassified affections of the eye has materially enlarged the group of ocular tuberculosis; and it is more than probable that closer attention to resisting and chronic forms of ocular inflammations, conditions which fail to respond to the usual methods may be found to be tubercular. In fact, the writing of this paper was stimulated largely by certain experiences which I have had with tuberculin during the last ten years. During this period it has been a part of my daily routine of practice, to employ tuberculin as a diagnostic and a therapeutic agent, and the results obtained have been increasingly gratifying as my experience in its use has grown.

TUBERCULIN AS A DIAGNOSTIC AGENT

Several phases of its use as a diagnostic agent are worthy of consideration.

(a) *Method*.—But two of the methods will receive consideration in this paper, namely, (1) the cutaneous, and (2) the sub-cutaneous. Of all the methods which have come into favor from time to time, these two alone have been found sufficiently accurate and dependable for daily use.

1. For the *cutaneous test*, which is best represented by the von Pirquet method, many physicians have shown a preference. It was thought for a time, that by this method one could carefully differentiate between an old or healed focus of disease and one which was active. It is exceedingly sensitive, and according

to those who are especially qualified to judge, it may be positive in individuals whose lesions, long since, have ceased to be active. It, however, is also positive in active lesions. It cannot, therefore, be depended on for this differentiation. In children it is nearly always positive and, therefore, may be misleading.

2. For some time past I have abandoned all but the *subcutaneous method* and this seems to be the tendency of a large number of men who use tuberculin. The subcutaneous method has a distinct advantage, first, in detecting active tuberculosis rather than healed foci of the disease. In the second place it furnishes us with a constitutional, a focal and local reaction. For the purpose of determining the character of an eye lesion, the focal test may be most important. The third advantage of the subcutaneous test lies in the fact that the diagnostic dose often acts therapeutically as well. Several marked illustrations of this advantage have recently been brought to my attention.

The disadvantages of the test need hardly be considered because any accurate test in medicine should be painstakingly applied and the action of tuberculin should be most carefully studied, preferably in a hospital. When hospital facilities are not available, an intelligent mother may be taught how to take and record a three-hour temperature.

The patient's temperature should be taken at stated intervals for four days before the test is applied to determine the presence or absence of an abnormal rise and in order to have a standard for the individual patient with which to compare any possible reaction after tuberculin is administered.

(b) *Preparations to Be Employed and Dosage.*—Old tuberculin is generally conceded to be the best preparation for diagnostic purposes. Remote or secondary lesions, such as are found in the eye, may be caused by any of the various forms of toxins elaborated by the tubercle bacilli in their activity in the primary focus or by their dead bodies. As old tuberculin contains practically all of these toxins, it is well adapted for testing purposes.

The profession is much at variance as to the dosage to be employed. In eye work it is important that the initial dose should be small. A focal reaction is the most convincing of testimony as to the character of the lesion, but it may also be a very serious one for the delicate eye structure. It is better judgment to begin with a small dose, 0.005 mg. for children and 0.05 in adults. If no reaction, either focal or constitutional, occurs, the dose may be doubled. Occasionally while making diagnostic tests with increas-

ing doses, instead of a reaction, a marked improvement in the local condition may occur. This improvement may be looked on as of as much diagnostic value as a positive reaction and the therapeutic administration may be instituted.

Occasionally I find a negative reaction with even large doses of old tuberculin, in cases which clinically have the ear marks of tuberculosis. A change in the preparation of the tuberculin from O. T. to T. R. in these cases will sometimes produce positive reactions. The reason for these occasional failures depends on the fact that the special antitoxin required is not present in old tuberculin or, if present, not in sufficient quantity to produce a reaction. Most tubercular eye lesions are caused by a toxemia—a tuberculoid, as the late Dr. Wendell Reber uniquely styled it. The toxemias of tubercle bacilli, whether due to the activities of the living or dead micro-organism, are varied and as old tuberculin is a filtered preparation, any particular antitoxin may not be present in sufficient quantity for clinical purposes. T. R. or any preparation of tuberculin which includes the bodies of the micro-organisms, on the other hand, may contain the desired antitoxin to produce results.

AS TO THE THERAPEUTIC USE OF TUBERCULIN IN EYE CONDITIONS

Ocular tuberculosis is perhaps the most ideal form of tuberculosis to yield to tuberculin when therapeutically administered. With few exceptions it is a secondary form of the disease. The bacillus can rarely be found in the lesion and furthermore tubercular processes cannot always be induced by animal experimentation. These two conditions are regarded by laboratory scientists as essential to unquestionably establish a given pathologic lesion as tubercular in origin. One hesitates to question the authority of the laboratory on its right to so dogmatic a conclusion. From a clinical and therapeutic standpoint, however, I am quite sure certain cases challenge its infallibility. A positive result is undoubtedly convincing, but negative findings do not necessarily destroy the value of clinical evidence. The tubercle bacilli have not been found in phlyctenules, so far as I have been able to learn, but no careful observer will place himself on record to the extent of denying any vital or cause-and-effect relation between tuberculosis and phlyctenulosis. In fact, the delicate eye structures are particularly susceptible to the action of toxins. For example, the behavior of interstitial keratitis, supposedly and probably in most instances syphilitic in origin, suggests a toxemia

rather than the actual presence of the micro-organism. It responds feebly, if it all, to arsphenamin (salvarsan) and allied preparations and with considerable uncertainty to mercury. Like phlyctenulosis, interstitial keratitis is associated with a latent stage of the disease.

These toxic types of ocular lesions, if I may so call them, are the ones which yield most readily to tuberculin. They, for the most part, are found in the anterior optic segment and the cornea is the favorite seat of activity. In addition to phlyctenular processes and intractable types of interstitial keratitis, a deeply seated corneal infiltration is the form most frequently found. The latter lesion consists in a small white area, 2 or 3 mm. in diameter without much ciliary involvement. Insidiously, however, a quiet iritis develops which later becomes active and a deep scleritis completes the picture. In the early stages I have observed these nodules disappear after the injection of several diagnostic doses of tuberculin. Even a small dose of tuberculin will sometimes cause a focal reaction as indicated by a sudden ciliary inflammation and a deep scleritis. One can therefore fully appreciate the dangers of too large a primary diagnostic dose as well as the necessity for small therapeutic doses.

DOSAGE

As a curative measure the initial dose should be 0.0001 mg. in adults and in children. If this causes a reaction either focally or in a rise in temperature, from five days to a week should elapse before a second dose is administered and if the reaction is marked a smaller amount than 0.0001 mg. should be given. In children the increase must of necessity be slow and it is wise policy to exercise the same care in adults. It is not necessary that huge doses be administered to accomplish results. In many instances a small dose is followed by a drop in the temperature rather than by a rise. When this can be accomplished it is ideal. The focal reaction, however, is the best guide as to when and how much to increase the amount to be given. In the deep corneal infiltrations, referred to a moment ago, a quantity of tuberculin even minutely in excess of the point of toleration, may be followed by a deep scleritis and iritis. The former condition is the more serious because of the greater time required before it is safe to increase the injection. After a few weeks of treatment one can readily determine the tolerance of the patient and thereby guard against excessive reactions.

Minute details are of the greatest importance throughout the treatment as a lack of care may not only lead to failure but to positively harmful results as well.

COMMENT

The citation of case histories will not add to the purpose of my paper. Tuberculin needs no defense. It is a product of admittedly great merit, and in harmony with the trend of modern medicine. It is simply my hope and purpose in this brief résumé of my experience with tuberculin in the last ten years, to add whatever of value this experience may contain, to the sum total of our knowledge of this very interesting subject.

The field is large and the opportunities are many. The technic, however, differs from that of the ordinary administration of tuberculin in pulmonary tuberculosis, for example; and this technic must be mastered in minutest detail in order to obtain results in the treatment of ocular tuberculosis.

DISCUSSION

DR. H. A. SMITH, Delta, Colo.: Dr. Peter has covered this field in general and in detail. I am delighted to hear it has been used over ten years. It is the only remedy that can be watched first, last and all the time. If you do not watch the eyes of your patients all the time you will fail in your results. It is an absolute and sure remedy. It clears up some cases that have been overlooked and clears them up more than any other remedy, not barring the Wassermann test. The injection hypodermically is the only way to use it. Other ways are liable to misinterpretation. I use a smaller dose than recommended here. Day by day you follow with the ophthalmoscope, and by giving an injection as small as one-millionth, you will find a little change around the edge of your lesion. If you do not, try a little more. As soon as you find it, you have the dose. It is the focal reaction. The least reaction is the time to carry your dosage. You do not want to set up reaction.

DR. L. N. GROSVENOR, Huron, S. D.: Five years ago I had a woman of 45 who had phlyctenule at several points about right corneal margin. This condition had been neglected for two months before she came to me. I found a severe irido-cyclitis with these persistent phlyctenules. Iris small and many synechiae. These gradually extended into the cornea, from each of these points, a dense semicircular opacity, small at first and increasing in size. These did not coalesce and cover the cornea as you would expect an interstitial or a sclerosing keratitis to do. The Wassermann test was negative, and the von Pirquet test positive. She was sent to the hospital and given a course of treatments with the von Ruck tubercular vaccine. In a short time this relieved the acute inflammatory conditions and later, when the opaque areas cleared up, then, and not till then, was it possible to clearly see the small tubercular nodules in the periphery of the iris; these undoubtedly also involved the ciliary body. Another series of tubercular vaccine treatments was necessary, about a year later, to clear up an incipient similar trouble in the left eye. There has been no active recurrence of the tubercular trouble in either eye for four years.

MAJOR W. H. LUEDDE, St. Louis: The conclusions of the essayist approving of the use of tuberculin in ocular lesions of tuberculous origin, fully coincides with my own experience. My personal observations in this line began at the Augenklinik at Kiel, Schleswig Holstein, in 1905, immediately after the Heidelberg Ophthalmic conference of that year, at which its use was so strongly advocated by von Hippel. The use of it was begun during my stay at the Rothschild Foundation d'Ophthalmologie in Paris in the following year, and I have since used it continuously in clinical and private practice. Recent investigations made by Dr. Greenfield Sluder and myself in regard to the direct etiology of these ocular lesions afford an explanation for some cases in which tuberculin proves a disappointment. We found with surprising frequency a focal reaction in the mucosa of the nose and accessory sinuses to accompany the focal reaction in the ocular lesions after test injections of tuberculin.

Theoretically it is much easier to assume that the tubercle bacillus or its toxins find access to ocular tissues through the close lymphatic and anatomic relations between the nasal and orbital cavities than that they should be carried to the eye from the lung or other constitutional foci. When the tuberculosis is scattered throughout the system as in the miliary type of the disease, the clinical picture locally and generally is very different from that ordinarily present in ocular tuberculosis. In fact, less than one half of the cases of ocular tuberculosis show pulmonary tuberculosis. These considerations and their relation to the frequent bilateral symmetrical character of the lesions in ocular tuberculosis are discussed in an article on "Ocular Tuberculosis of Nasal Origin," published in the *Annals of Ophthalmology*, January, 1916. Therapeutic proof of the correctness of our theory was supplied in two cases where previously tuberculin had checked the ocular disease but failed utterly during a serious relapse several years later. In one of these young women the choroid was involved, and in the other the cornea; both were bilateral. A radical operation by Dr. Sluder for the perfect drainage of the ethmoid and sphenoid (right and left) resulted in effectively checking the disease. During three years subsequently not only has there been no relapse but the ocular condition has undergone marked improvement.

DR. M. FEINGOLD, New Orleans: The report of the two cases by Dr. Luedde contains a great deal more than is visible on the surface. We so often meet with uveal affections in both eyes of our patients and it has always appeared difficult to explain the bilateral manifestations of the disease. But if foci in the ethmoidal or sphenoidal sinuses are found more often in these cases, it will be comparatively easy to assume, that from these centrally located foci metastatic extension occurred into both eyes by the way of the blood or lymph vessels.

MAJOR LUEDDE: That is just in accordance with our experience.

THE CHAIR: Those of us who live in the West and have occasion to see tuberculosis in all its manifestations, surely have come to learn that ocular lesions require investigation so far as tuberculosis is concerned. It is only just now receiving the attention it is entitled to receive.

LITERATURE OF OUR SPECIAL WORK:

HOW TO PRESERVE FOR ITS BEST USE

LORENZO N. GROSVENOR, M.D.

HURON, S. D.

One day some twenty-five years ago, my good father came into his office and found a lady waiting for him. While waiting she had perused one of the medical journals on the table. She at once remarked: "Doctor, did you read this wonderful article by a doctor who cures all his cases of scarlet fever? My! but he must be a wonderful doctor."

Father looked over the article for a minute, then laughing turned to the lady and said: "Notice that this writer has indeed cured every case of scarlet fever he ever had, but note, too, that this fellow just graduated in medicine only a few months ago and has rushed into print with a flamboyant write up of curing the only two cases of scarlet he ever had. He will know better in the years to come."

There is, in Chicago, a physician, yes, in our special line, too; a full-fledged professor at that. This man was in our eye department at Rush Medical College for a time, but when our dear old head professor found out what an illiterate fellow he was, he promptly dropped him from the department. Within a few months this same man bought a full professorship in one of the minor medical schools in Chicago. Later he blossomed out in the special society with a paper describing a "wonderful discovery about the tonsils." Well the paper was respectfully received, but all, with one accord, could not but feel chagrined that such a paper should be presented to a society noted for its scientific programs. The professor in his monumental gall and unblushing cheek and absolute ignorance of textbook and current literature of his subject, had rushed into print with a wonderful discovery, whereas, if he had been informed on the literature of the subject he would not have made such a foolish move.

When you are asked for a paper for the Society do *not* go and copy a verbose textbook article and pawn it off as your own ideas, for most of us catch on quickly. How to avoid such foolish moves is the purpose of this paper.

One thing that I miss more than anything else away from dear old Chicago, is the splendid eye, ear, nose and throat department of the Crerar Library in the old Field Annex Building. Very few of the larger cities have such a fine, large and complete library of books and journals of our lines. Well, what shall we do about it?

STUDY THE CURRENT LITERATURE

Textbooks.—One should buy at least one best book of his special line of work each year. Having it, he should read and study it thoroughly.

Journals.—At least be a subscriber to the one best eye journal and the best ear, nose and throat one. When the journal comes read and study it carefully. Do not throw it into the corner and forget it. When read, file it away carefully and when the volume is complete bind it with its index so it becomes useful.

You undoubtedly take several general medical journals. Scattered through these are many excellent articles on subjects of our special lines. Tear these articles out carefully, read them, fold once and file in the drawer for a time. Also read and save all reprints in our lines. These are to be read and placed with articles torn out, and, when a bunch has accumulated, at your leisure or that of your office girl, catalogue them, library fashion, and they instantly are changed from useless lumber into most useful references.

Classification.—For this, there is the well established and universally used Melvil Dewey "Library Classification." Years ago (1906) I modified this to fit our special lines of eye, ear, nose and throat. I use 617.7 for eye, 617.8 for ear, 617.9 for nose and throat; expanding each as necessary. This brings all papers on the same subject together, but you note that there are various authors on the same subject. How shall we differentiate? You should get out your Cutter's "Author's Alphabet Classification." Under subject title number, the author class number should be placed. On the reprints and papers torn out you can write under and to the right of title the subject number and under this the author number. Do this on all the articles to be filed referring to the tables for subject and author classification numbers. Now each article should be placed in an envelope large enough to hold them. I use one the size of 6 $\frac{1}{8}$ by 9 $\frac{1}{4}$ inches, "Victor 1331, No. 2," portfolio manilla. Your druggist or stationer can secure them from McClurg and Company of Chicago for about \$3 per thousand.

On the envelope, with the typewriter or pen, one-quarter inch from the top and 2 inches from the left end of the envelope, you should write the classification subject number; then one-half inch to the right, title with keynote word is to be written first followed by the rest of the title as printed. Under the subject number place the author number, followed by the name of the author, last name first. The journal from which the article came, with date of the journal, should be noted.

These filled envelopes should then be filed in drawers of the filing cabinet in the order of their classification numbers. I am using the Baker-Vawter No. 400 steel storage units in sets of six high, costing about \$20 for the set. These drawers are fine for filing catalogues, invoices, etc.

Titles.—A word here to all of you: when you send in a title to the secretary you should remember that it will be catalogued by Uncle Sam in the Surgeon-General's Library Catalogue, by the Index Rerum folks—by Jackson, if an eye subject; by Beck and Goldstein if an ear, nose and throat subject. If the title is first presented properly by the author and tersely worded, giving keynote first, it will be powerfully useful to the other fellow, when he wants everything published on that subject. So many articles are lost, and what might be a most useful article is buried by an obscurely worded title when copied into the index or catalogue.

Clippings.—In the medical journals there are abstracts of articles from other journals. These should be glanced over for eye, ear, nose and throat titles and, if found, they should be torn out and saved. When a bunch has accumulated they should be trimmed and sorted by subjects, pasted on 5 by 8 cards and filed, library fashion, for future reference.

You should save pictures of celebrated men in our line, paste them on the cards with the life history, if available. Again, the picture of an author should be pasted on the title page of his book, if you have one.

Your special journals should be filed away carefully and bound at the end of the year. If there is no bindery in town, you should bind them yourself.

Now then, why have I perpetrated such agony on you as this paper? Often you run across cases that pester the life out of you to know what next to do. Not only should you get out your textbooks but you should go to your filing case and collect

all the envelopes on the subject, sit down and work out your salvation. The index catalogues of Jackson and Beck and Goldstein help wonderfully in our work.

Again, if you are asked for a paper for this or any other society you have a lot of reference literature at your command and can do yourself justice. One of the best informed eye men in Chicago once explained to me that, in writing papers, one should exhaust the literature on the subject by careful research work, digest it, and write such a paper that will command the respect and commendation of the best men and the other fellows will quickly fall in. One needs to be in good grace with himself and his fellows of his special line.

DISCUSSION

DR. EDWARD JACKSON, Denver: I want to call attention to the fact that the working over of the literature requires a certain amount of time and attention paid to it; an immense amount, whether we follow this particular system of Dr. Grosvenor's or one of our own. Unless we follow some system we either throw away journals, or read them as we do the newspapers with the idea of picking out what is interesting.

One word with reference to titles. Of late they have been better than in the past. The purpose of the title is to tell what is in the paper. If it does not tell that it is a failure. The title that merely tells that the doctor has had some cases should be ruled out by the editor. The title should tell us what the paper refers to. I have some feeling on this because I have gone so far in ophthalmic literature that when the title does not tell the subject I do not copy it into ophthalmic literature. I try not to repeat the errors of a man who writes a paper and gives it a long title.

The first textbook we read on any particular subject is perhaps more valuable to us than any amount of literature we will ever read afterward on that subject; and the proportion of what is new to us afterward decreases with each book we read. Nevertheless, perhaps once in ten years, comes a book we must read and be familiar with, and the later editions of such books as Weeks and deSchweinitz, etc., are much more valuable than the previous editions, as they have been gradually developed. Journals are usually more valuable than textbooks. One more phase is the Encyclopedia of Ophthalmology. Literature is valuable for reference, and if it is arranged systematically it is all the more valuable. This literature is valuable, not for what we can remember, but for what we can find when we want to look up a particular subject.

DR. H. V. WÜRDEMAN, Seattle: I have worked for thirty years with essays and editorials in the same line as Dr. Jackson, but even yet find in our wonderful development of the *Journal of Ophthalmology* we have less than 25 per cent. of the alleged ophthalmologists of America as subscribers, and I want every one here to be an active propagandist for the journal. It belongs to each and every one of us. We can send to it questions or anything worthy of publication, and we want the men to contribute, especially the new men. We older men are losing our bright ideas, but every one runs across something or works out something of value. Papers before medical societies should be about one subject and be restricted to one idea, and that should be the title of the paper. One

new idea is worth more than any syllabus of the literature you can bring us. The citation of case histories, as that of Dr. Reeve, is unusual and well worthy of publication. We do not want to know who referred the case to you or have other extraneous matter included in the paper unless it refers directly on the subject. Make your papers short; as Dr. Wood said to me when I asked him what I should talk about at an after-dinner speech, "Talk about a minute." As a rule, a ten-minute paper is more valuable than a thirty-minute one.

DR. M. FEINGOLD, New Orleans: A word of recommendation. All those in charge of compiling medical literature, ophthalmic or otherwise, should see that all that is brought out in the discussion should be properly cross-indexed. This has until now been sadly neglected on all sides. In consequence of it many valuable cases deposited in the literature are lost and the labor of looking up the literature is thereby considerably increased. Proper cross-indexing will also act as an incentive to the beginner who is too timid to report his cases in separate papers, but who will relate his experiences in the discussion.

THE COMFORT TEST AS A METHOD OF REFRACTION WITH DEMONSTRATION OF A SPECIAL SET OF LENSES AND FRAMES

PETER POTTER, M.D.

BUTTE, MONT.

The fundamental idea of this paper is that the comfort of the patient is a reliable and satisfactory guide in the fitting of lenses. So many patients complain of the discomfort and inconvenience of having a mydriatic used in the eyes when they are to be tested for lenses, that I have practically discontinued its use, and for several years have been using a method which I shall describe as the comfort method. The results have been so uniformly satisfactory that I feel justified in bringing the subject to your attention at this time. If we were more considerate of the comfort of our patients a much larger number of people would be cared for by competent oculists and a fewer number by incompetent opticians.

What is presented in this paper is not a new method, rather a refinement of old methods which adds to the ease and comfort of both the oculist and the patient. The original idea was obtained from Dr. John Green, Sr., of St. Louis. Several years ago, in fact while I was still a premedical student in the University of Missouri, my room-mate was refracted by him by a method different from anything I could find described. This made a deep impression on me so that after I had been doing refracting for a short time, and found that so many people objected to the use of a mydriatic I began to use Dr. Green's method. If a man of Dr. Green's reputation could run the risk of going counter to accepted ideas, I could do likewise, if I could thereby get more satisfactory results. I soon found that I was able to get at least as good results without the cycloplegic as with it, and from this kind of a beginning, I have gradually developed the method which I depend on almost exclusively at present.

PRINCIPLES OF THE COMFORT TEST

The first part of the method is a modification of the ordinary fogging system; the second part a modification of Dr. Green's method.

Fogging may be done by either the upward or downward method, that is, by beginning with weak plus lenses and increasing their strength until the vision is fogged to the desired point, or by fogging the vision markedly at first by the use of a strong plus sphere, plus 7, and reducing the strength of this lens until the vision is fogged the desired amount. I use the downward fogging method entirely, because my experience leads me to believe that this gives more uniformly satisfactory results. Instead of beginning with a +7, then substituting a +6.50, then a +6, and so on as is usually done, I prefer to leave the +7 in front of the eye continually and reduce the strength by using minus lenses with -0.50 intervals until the strength is reduced to the point at which the vision is properly blurred.

The objection to using the upward method of fogging is that the patient is continually using the accommodation—the very thing the test aims to overcome—while with the downward method the strong plus sphere causes a relaxation of the accommodation much quicker, and the relaxation can be maintained to the end of the test.

The details of the method have been described in a short article on the subject, published in the *Annals of Ophthalmology* for July, 1917, from which the following description of an ordinary test is taken:

DETAILS OF THE METHOD

After the patient's history has been taken, he is placed before the test cabinet and the phorometer is adjusted so that he looks exactly through the centers of the lens holders, with the head in as natural and as comfortable a position as is possible; the left eye is covered and the best possible naked eye vision of the right eye obtained; he is encouraged to read the smallest letters possible, and the mistakes and amount of apparent effort are noted; a toric +7 sphere is placed in the back cell of the phorometer as close to the eye as is possible without the lashes touching the lens¹; the patient is advised to keep the eye as quiet as possible and to make no effort whatever in trying to overcome the blurred vision. When the eye has had time to accustom itself to the blurred vision, a -0.50 sphere is placed in the front cell of the phorometer. After a few seconds the -0.50 sphere is removed and a -1 put in in its stead. This process is repeated, increasing the strength of the minus lens by 0.50, until the patient is able to read 20/40. Sufficient time is taken between the changes to allow the muscle of accommodation to relax and to become entirely adjusted to the combination of lenses in front of the eye. If the patient

1. A toric sphere is used for two purposes: (1) in order to get the lens close to the eye, and (2) to reduce the space between this lens and the lens to be placed in the front cell of the phorometer.

seems to be nervous or for any reason unable to relax the accommodation, an extra amount of time is allowed between changes, and the patient's attention distracted from the test by engaging him in conversation. When he is able to read 20/40 or a little better, never more than 20/30, the minus lens is removed and a plus lens is put in its place, the strength of which is 0.25 or 0.50 more than the difference between the minus lens in the front cell and the +7 in the back cell; that is, if there is a -6 in the front cell, the difference would be +1. If the patient is able to read 20/40 with this combination, a +1.25 is placed in the front cell; if he is able to read 20/30, a +1.50 is used; the +7 is removed, and if the patient reads as well or better with the single sphere than he did with the combination, a sphere 0.25 or 0.50 stronger than the one in the front cell is then placed in the back cell and the front one removed. The same process is then gone through with the left eye.

The reason for substituting a sphere stronger than the combination of the +7 and the minus lens is that with the two lenses we have four strongly curved surfaces and an interspace. This combination interferes with vision more than the two weakly curved surfaces of the equivalent single lens, the result being that the patient will be able to read one or more lines on the chart with the equivalent lens than with the combination. This increased strength of lens is used on account of the fact that it is not advisable to have the patient read below 20/30 at this stage of the test, because the test for astigmatism is best done with the vision fogged to the point that only one or two sets of lines are distinct rather than that all should be fairly distinct and only one or two showing some blur.

The important part of the test is to determine the exact amount of astigmatism present, and for this purpose the following method of testing is an improvement over that ordinarily described by men using the fogging method. With the patient unable to read more than 20/30, the astigmatic chart is placed before him and he is asked to decide which set of lines is most distinct. If he decides quickly it is evident that there is considerable astigmatism present, and one may begin with a fairly strong cylinder; if he hesitates and is unable to decide definitely which is the more distinct, it is probable that the astigmatism is not very marked and that only a weak cylinder is required. A minus cylinder is placed in front of the sphere, with its axis opposite to the direction of the most distinct lines; if this lens does not make the lines equally distinct in all directions, then minus cylinders of increased strength are used until the lines of the chart are equally distinct in all directions. However, if the minus cylinder required is sufficient to reduce the strength of the combination enough to bring the vision down to 20/25 or better, a stronger sphere is substituted for the one in the back cell of the phorometer, and the process of determining the proper cylinder is then continued, always keeping the vision fogged to 20/25 or

poorer.² After the minus cylinder which seems to equalize the lines has been selected, a -0.25 cylinder is held in front of the combination; first, in the same plane as the other cylinder, then, at right angles to it. If this does not accentuate the corresponding set of lines when held in either direction, the original cylinder is not correct and the test must be continued until this final test can be applied. This last test may be verified with a $+0.25$ cylinder. With the strength of the cylinder satisfactory, the correct axis is determined by rotating the cylinder until there is a difference in the distinctness of the lines. After noting the position of the axis when the patient is first conscious of the change, the cylinder is then rotated in the other direction until the lines are again unlike. The mean of these positions is usually the correct axis. This may be verified by having the patient rotate the cylinder and place it in the best position. Usually the position found by one method practically coincides with that found by the other. Experience indicates that corrections need not be made closer than quarters, either with spheres or cylinders, or by less than 5 degree intervals, hence a large amount of detail work is avoided without any appreciable loss to the patient.

It occasionally happens that the patient will select as the most distinct set of lines those opposite to the ones which really should be most distinct. This occurs when the eye is not sufficiently fogged or possibly too much fogged. The error can be recognized before the test is completed and the proper correction substituted. If we assume that the conoid of Sturm is the correct figure to illustrate the relationship of the rays of light as focused by an astigmatic eye, there will be little difficulty in understanding this apparent change of axis.³ Experience seems to indicate that if the vision is kept fogged between 20/25 and 20/40, the focal interval is kept in front of the retina, yet not far enough in front of it to cause too much blurring of all parts of the chart. This is the reason for using a sufficiently strong sphere in the back cell of the phorometer to keep the vision fogged to 20/25 while the minus cylinders are being used.

This amount of fogging, by keeping both focal lines in front of the retina, puts the eye into a state of artificial compound myopic astigmatism, and no amount of accommodation can improve the vision. In this condition the lines on the astigmatic chart corresponding to the posterior focal lines will be the more distinct ones, and will remain so as long as the posterior focal line is the one nearer the retina. If now the anterior focal line is thrown backward toward the retina by means of minus cylinders placed in front of the sphere, the focal interval can be

2. This change of sphere is to be made in such a way that at no time is the eye left without a plus lens at least as strong, if not stronger, than the one found by the test up to that time. To accomplish this a plus sphere stronger than the one in the back cell of the phorometer is put in front of the minus cylinder. With this second sphere in place the one in the back cell is removed and the desired sphere put in its stead. The sphere in front of the cylinder is now removed and the test for astigmatism continued.

3. Refer to Fuchs' Fig. 384 (Text Book of Ophthalmology, third English edition) or similar figures in other textbooks for graphic illustrations of this geometric figure.

obliterated, the astigmatism neutralized, and the eye thus made artificially myopic. If, however, too strong minus cylinders are used, the anterior focal line will be thrown behind the posterior one and the eye again becomes astigmatic, but in the opposite direction. Because the original anterior focal line is now the one closer to the retina, an opposite set of lines will be the more distinct.

An increase in the strength of the minus cylinder will increase the difference in distinctness of lines on the chart until this focal line has reached the retina, when a further increase of the minus cylinder seems to equalize the lines again. At this stage careful observation will bring out the fact that the chart is becoming blurred, that certain letters are not read as correctly as before, and that the patient is making evident effort at accommodation. The eye is now in a condition of artificial mixed astigmatism, and the accommodation can, and does, materially affect the results obtained by the test.

If the eye is so fogged that the posterior focal line is behind the retina, but nearer the retina than the anterior focal line, a certain set of lines will be the more distinct, but as the minus cylinders are added the anterior focal line comes to be the one nearer the retina, so that an opposite set of lines becomes the distinct ones, and this without having fully corrected the astigmatism. The natural tendency under such circumstances would be to reduce the strength of the minus cylinder, while in fact it should be increased in order to bring the two focal lines together, neutralize the astigmatism, and thus convert the eye into an artificial condition of simple hyperopia. This error is to be suspected when the same combination of lenses gives varying results, and especially if the patient seems to be making undue effort to see. The suspicion is confirmed if by increasing the strength of the sphere in the back cell of the phorometer the patient is able to read as well as with the weaker lens, without the evident effort of accommodation, and the lines of the chart can be equalized more satisfactorily by a stronger minus cylinder.

Sometimes this error is not recognized until the near test is applied, when it is found that the patient will accept considerable increase of sphere, showing that he has been using too much accommodation, and the lines of the chart can be equalized more satisfactorily by a stronger minus cylinder. All such tests must be repeated under proper conditions of fogging, in order to be certain of the correction for astigmatism.

If the eye is so strongly fogged that all lines on the astigmatic chart are badly blurred, the patient may not be able to discern that one set of lines is more distinct than the others, but in his desire to assist in the examination he may accommodate strongly and at the same time, as a part of the same muscular effort, contract the pupil and pull the eyelids together. This results in a reduced and more distinct image on the retina, and may change the curvature of the cornea enough to bring a set

of the blurred lines to a little better focus. This change in shape of the eyeball may convert the eye into an astigmatic condition opposite to its natural astigmatic state. Minus cylinders used as directed in the foregoing will exaggerate rather than correct this astigmatism, hence an apparent change of axis will take place before the test is completed.

The same method is used with the other eye, using as much care in the fogging and in determining the cylinder, because it is not uncommon to find that the two eyes differ so materially that no opinion of what should be found for the second eye can be formed from what has been found for the first eye.

The last part of the test is to determine the strength of sphere necessary. The astigmatic correction is always determined, as stated above, with the eye so fogged that the vision is not better than 20/25 at any stage of the test. After determining the strength of cylinder under these conditions, the sphere is gradually cut down until the vision is a little less than 20/20, or such that 20/20 is obtained with a —0.25 sphere in front of the other combination. That is, the strength of lens to be left in front of the eye should be at least 0.25 stronger than that which gives 20/20 with the single eye. After both eyes have been brought separately to this stage, it is found that the two eyes together have 20/20 vision with the lenses 0.25 stronger than that which gave 20/20 vision with each eye alone.

OTHER CONTRIBUTIONS ON THE SUBJECT

Major W. B. Lancaster presented a very valuable paper to this Academy at its twentieth meeting, in 1915, on "Subjective Tests for Astigmatism" which together with the foregoing quotations simplifies many of the difficult problems met in correcting astigmatism without a cycloplegic, and shows why men who have no inclination to pay attention to minute details condemn such a method before having given it a fair trial.

RECENT OBSERVATIONS OF THE AUTHOR

During the two years since my paper was written I have had the opportunity to retest, for one reason or another, many cases that had been fitted from one to ten years ago, and I desire to say that I am more firmly convinced now than at that time that the results are just as satisfactory as by any other method. In the large majority of these cases I found that the corrections for astigmatism were just the same as at the previous test, and that if any change was necessary it was usually to increase the strength of sphere. This increase was made necessary either by increase in age, that is, nearing the presbyopic stage, or because the patient

had changed occupation and doing more close work than when the original lenses were prescribed. Occasionally he was doing less close work so that weaker lenses were more acceptable.

The fogging must be followed immediately by the near vision test. The near vision should be tested as carefully as the distance, because it often shows inaccuracies in the distance test and thus becomes the first check on the test. Also most cases of eye discomfort are from close work and not from distance hence an accurate near test is an essential part of the process.

These constitute the preliminary part of the test, but were they the only portion, I certainly would not appear before you, because a large percentage of glasses so fitted would be unsatisfactory. In order to guard against this I have had made a set of round compound lenses, and a set of light trial frames. By this means I am able to determine if the lenses as found by the preliminary test are comfortable. This is the portion of the method which I desire to bring to your attention, and is the only part for which any originality can be claimed. Since the lenses are round, the axis of the cylinder can be placed at any desired angle, thus making each lens serve theoretically for 180 prescriptions, and practically for at least 36. The strength of the lens and the axis of the cylinder are etched on the lens in such a position as to be easily read, and yet not in any way interfering with the use of the lens. The strength is indicated by numbers, the sphere first, and the cylinder second, as $+0.50 +0.75$; $-1 -0.50$; $-0.25 +1.25$ meaning $+0.50 S \subset +0.75 \text{ cyl.}$, etc. The position of the axis of the cylinder is indicated by a short etched line at each end of the axis.

SELECTING THE FRAMES

The frames, about 30 in number, are so chosen as to duplicate practically any frame measurements, so far as pupillary distances and bridge dimensions are concerned. These frames are very light and comfortable, and when such a frame and pair of lenses are being used, the patient is little or no more annoyed with the set than if it were his finished prescription. Experience indicates that less than 200 pairs will duplicate practically every prescription to be written in the ordinary routine office practice.⁴ Even a much smaller number, if carefully selected and confined to single lenses, in the less frequently used combinations, will suffice, but

⁴. Two lenses may be fastened together with adhesive in order to obtain combinations not in the set.

the satisfaction of being able to duplicate any desired prescription will warrant a complete set.

The lenses are made from ordinary lens stock, and should be just as comfortable and serviceable when in a properly selected frame as if they were the final prescription. Care, of course, must be exercised in selecting the frame, because of the discomfort from improperly mounted lenses. The frames will also accept the lenses of the ordinary trial case, so that these may be used where simple spheres or cylinders are needed and thus keep the lenses in the special set down to a reasonably small number.

When this part of the test has been completed the prescription is made up from the set and the patient wears it in the office or at home until its accuracy can be determined by his comfort. The lenses are to be changed as the symptoms indicate or as further tests require until a combination is found which the patient can wear with comfort for his usual work, for as long a time as he will use his finished prescription for this same kind of work. Anything short of such a result should not be accepted.

COMMENT

It should be distinctly understood that I do not claim to be able to fit satisfactorily every patient who calls on me, but I do claim to be able to fit satisfactorily a larger proportion of cases with less annoyance, and in less time, both to myself and to the patient, than is customary when a mydriatic is used. If, after repeated tests, we do not get a satisfactory lens, the patient should be discharged, because experience has proven beyond a doubt that a lens which is not comfortable in the office test will not be comfortable at home, and its prescription is not justified.

These are the cases to be tested under a mydriatic, if a mydriatic is to be used at any time; however, I am frank to say that I have practically discontinued testing such cases by that method, because I rarely get any more satisfactory results in that way than by the comfort test. These are usually the cases in which the eye symptoms are only local manifestations of some systemic disease, and general examination and treatment are necessary to relieve the condition. Such cases are referred to a diagnostician, and the usual result is that sufficient is found to account for the symptoms, and glasses are not prescribed, or, if prescribed, are for temporary use only.

Cases of weakened accommodation are not to be fitted with the full strength of lens necessary for comfort during long continued use, but rather the weakest lens possible for the amount

of work which it is absolutely necessary for the patient to do, and then with the injunction that the eyes are not to be used beyond the point of beginning discomfort without a few moments of rest. Such eyes will often do several hours of close work each day without much fatigue if allowed frequent short periods of rest. No amount of testing under a cycloplegic will determine the proper lens in such cases. The patient's comfort under the given conditions should be the guide.

REASONS FOR THE SUCCESS OF THE TEST

The reason why cases tested by the comfort test are as satisfactory as those tested under a mydriatic is obvious. With the comfort test the eye is in its natural condition and under as nearly the usual surroundings as is possible. The pupil is of its normal size and shape and admits the same amount of light it will admit when the lenses are being used at home. The retina and the muscle of accommodation receive and respond to stimuli as they will after the glasses are prescribed. The curvature and sclerosis of the periphery of the cornea and of the crystalline lens may not be the same as that of the central portion. This is true more particularly of middle-aged patients, in whom an irregular sclerosis is often present, so that the lens found from an examination wherein the entire cornea and lens are a part of the refractive system may be different from one found in which the pupil is of its normal size and shape and only the ordinarily used portion of the cornea and lens enter into the refractive system.

When the total amount of refractive error has been determined under a mydriatic, there still remains the uncertain factor of the effort which the muscle of accommodation will make after it has recovered from the effect of the drug. This factor varies with individuals, and with the circumstances under which the eyes are to be used.

This factor must be guessed at if the lenses are to be fitted at once, and the prescription will not be any more nearly accurate than if fitted by the ordinary fogging method. Lenses so fitted often must be changed one or more times before the patient can wear them with comfort; in other words, the comfort test is applied at home at the expense of more time and annoyance on the part of both patient and oculist. After a few such changes, the patient realizes that the oculist is guessing at the prescription, and soon loses confidence in the doctor's ability and integrity.

If the lenses are not fitted immediately after the test under the cycloplegic, but are determined by a postcycloplegic test, the main part of the cycloplegic test is lost, and the method again approximates the comfort test, because the final lens is the one which the eye will accept, regardless of what has been found under the cycloplegic. Again there is much inconvenience and loss of time on the part of the patient, with no better results on the part of the oculist.

The amount of time required for the comfort test is no more on the part of the oculist, and much less on the part of the patient, than when the test is made under a mydriatic, and most patients are much better satisfied when they do not have to go several days with their pupils dilated and muscles of accommodation inactive.

USE OF THE TEST IN MYOPIC CASES

So far I have referred only to various forms of hyperopic conditions. The discussion would not be complete without referring to the testing of myopic cases. In myopia, the eye is naturally in the same state as that into which the hyperopic eye is placed during the fogging test. In those myopic cases in which the vision is poorer than 20/40, minus spheres are to be used until the vision is nearly 20/25. When this vision is obtained, the remainder of the test is the same as with artificial myopia. If the natural vision is not poorer than 20/25, it is well to fog the case as if it were one of hyperopia, since the apparent myopia may be ciliary spasms. In cases of low myopia minus lenses are not to be prescribed until after they are thoroughly tested by the comfort test, and are proven absolutely to be myopic. It is not uncommon for the comfort test to take a case out of the myopic class and put it into the hyperopic class, so that it is necessary to be extremely cautious in prescribing weak minus lenses. The comfort test is the most reliable guide which I have found in these cases.

PRESCRIBING FOR SPECIAL CASES

When an increased strength of lens for close work is necessary it is often difficult to determine which will be the more comfortable, a bifocal set or two separate pairs of glasses. This question is readily settled by the patient when it is possible for him to compare in trial frames the exact strength full-sized reading glasses with the exact strength bifocals. To accomplish this, bifocal attachments have been made, and with these the

patient is able to determine very satisfactorily the amount of annoyance which the reading scales will give, because these approximate the final prescription so closely that the patient is able to recognize at once the advantages and disadvantages of bifocal lenses.

This set of lenses was made for me by the American Optical Company, and I wish to acknowledge their courtesy and assistance in the matter, for without their mechanical skill and patience, I would not have been able to develop the set to its present condition. This small set which I have with me for demonstration are torics, while my original set and the one with which I have done my work are flats. I shall be pleased to demonstrate the lenses and the method to any one sufficiently interested to spare the time for that purpose.

DISCUSSION

DR. JOHN GREEN, JR., St. Louis: We have to thank Dr. Potter for recalling to our minds the advantages of a very careful estimate of the dynamic refraction. The fogging which, as Dr. Potter has correctly stated, should be "downward," should be sufficiently great in the beginning to throw the entire focal interval in front of the retina. The strength of the sphere is gradually reduced until one or more lines of the astigmatic chart emerge clear and black. Only then does one begin to work with cylinders. I make a careful estimate of the dynamic refraction in every case, because I am convinced that in the majority of cases, a prescription based on the test without cycloplegia is the one which will give the most comfort. I have a long dark corridor at the end of which are my test letters, which are illuminated by mazda lights. The sphere is reduced to make vision equal 6/25 or 6/20. At the point where the eye can select as best one or more lines of the astigmatic chart, I begin to use minus cylinders and use successive strengths until the cross-lines are equalized. At this stage I find that vision has been improved but not to the best. The spheres are then gradually reduced, making a retest with cylinders with each spherical change.

As to the near vision tests, I have the patient use in reading in the office the combination which I have found to be satisfactory. I often prolong the reading test for one and one-half to two hours. Dr. Potter's sphero-cylinders, which the patient may take home and try, very perfectly represent the finished product and enable conclusions to be drawn which could not be arrived at in any other way.

If our trial lenses were made plano-convex and plano-concave, and mounted in thin flanged rims, they could be combined in a single cell trial frame—with plane surfaces in apposition, thus closely approximating the finished product of the optician. My father used always to insist on this point and his trial lenses were made and mounted as described.

I believe the fogging method is inapplicable in certain cases. I would not refract a child with active accommodation without atropin. Many a person with low hyperopic astigmatism is wearing weak minus lenses, not all of which were prescribed by refracting opticians. Many a myope has been given a higher minus glass than he should have. In mixed astigmatism it is almost impossible to make an accurate measurement without a cycloplegic. I believe that certain presbyopes,

with overactive ciliary muscles, cannot be satisfactorily fitted without a knowledge of the static refraction. Sufferers from painful accommodation require atropinization for long periods.

DR. JOSEPH LICHTENBERG, Kansas City: These frames of Dr. Potter's are very ingenious. They are exceedingly light. The American Optical Company makes a small set that has served me well, and I have never used these of Dr. Potter's. This will be beneficial in those cases in which the patients will not permit you to use a mydriatic. Second, those cases in which there is a slight suspicion of a glaucomatous tendency, and third, in people approaching the age of presbyopia. I cannot see how he gets an accurate test, although he tells me he does. From my own experience I do not think it necessary to often use the astigmatic charts. We rely on the visual acuity in most cases. This is an inaccurate method of the optometrist. I have been working on the draft board and on the advisory board, and in all these cases I have been astonished at the close approximation we get by having the patient in a dark room look at the distant object in estimating refraction with the retinoscope. Where we have a marked esophoria we simply must use a mydriatic, and nothing short of atropin will do.

DR. H. V. WÜRDEMANN, Seattle, Wash.: I hold that static refraction is necessary for the estimate of a prescription of lenses which will give comfort to the patient. I do not prescribe the exact measurement of the eye, but by the measurement under a cycloplegic we can work it out properly. It matters not in the majority of cases whether you give a plus 75, a plus 50 or a plus 90. These small differences make no difference to the patient. In the young person you correct a hyperope to a diopter or a diopter and a half. We have to prescribe one-third of their hyperopic estimate. I hold that the only correct method of measuring an eye is by the objective method. What the patient says does not matter. What he says should be corroborated. This contribution is valuable in saving us a great deal of time. None of the gentlemen present have ever produced a case of acute glaucoma by the use of atropin. I have never seen it. Homatropin has never produced such a case, and that is what I stay by. I want to say to you that most of us educated in the objective method of refraction, as most of us were thirty years ago, will want to use the cycloplegic for the measurement of the eye.

DR. J. A. L. BRADFIELD, La Crosse, Wis.: Exact refraction requires a knowledge of both static and dynamic refraction which can only be acquired in many cases by use of a mydriatic followed by a thorough test without. In the normal eye retinoscopy under a thorough mydriatic gives us a perfect dynamic distant refraction, but in cases of abnormal corneal conditions it may go far wrong, as in small corneal opacities and irregular astigmatism where the rays of light pass through an entirely different part of the cornea when the pupil is dilated to what it does in normal refraction.

In presbyopia there is occasionally a difference in the accommodation of the two eyes requiring a difference in the strength of lenses required for near use. Owing to the contraction of pupils for an accommodation the axis for far and near vision in high degrees of astigmatism requires a different axis for distance and near. All of which require a thorough post-mydriatic examination.

For the novice mydriatics are absolutely essential to good refraction in the young. The experienced refractionists will determine very accurately the proper glasses by the comfort test.

The eyeball is not a fixed sphere, but a living dynamic organ which must be considered after all means have been used to accomplish this purpose and the refractionist who depends entirely on retinoscopy in adjusting lenses will go as far or further wrong than the one who depends on the comfort test in all cases.

DR. H. B. YOUNG, Burlington, Iowa: When I began refracting, thirty-five or forty years ago, there was nothing but atropin for mydriasis and cycloplegia. I have my records of those days, and occasionally an old patient comes in to see if I can do better work now. More than once I have been astonished at the accuracy of my early work; and I charge it up to the fact that I used a real cycloplegic. I think that the report presented here by Dr. Potter is valuable in one sense and dangerous in another; the latter, because he has attempted to advise without knowing the real condition of the patient. Personally, I must know, first, all that I possibly can. If the patient does not want to do what I deem necessary in this respect, I promptly say that I am not a complacent doctor; the work is to be done in my way or not at all by me. In recent years a method that has appealed to me as a time saver and a reducer of discomfort to the patient, is that after going over the preliminary subjective tests and getting a line on the probabilities, the cycloplegic is put in one eye only. The binocular test which follows the monocular will generally show how much relaxation of accommodation can be depended on; and, if a partial correction is given, the patient is given advice to return for full correction. The difference between the acuteness of vision in the dilated and undilated pupil may be largely eliminated by using a modified pin-hole disk, opening not above 5 mm., before the one eye or both eyes.

DR. H. N. BLUM, New Orleans: In Dr. Feingold's clinic and in my own clinic we test eyes usually with homatropin, except in young children with strabismus or in those with ciliary spasm. We use 1 drop of homatropin in each eye and repeat in twenty minutes, a 2 per cent. solution, instilled carefully, having the patient look up and down so that it may be distributed, and we get as good results as with 5 or 6 drops used otherwise. We depend almost entirely on the objective test. We use the retinoscope carefully. Unless we have reason to believe that all the trouble has not been developed, we prescribe lenses based on that examination. If we have reason to doubt the accuracy of the test, we have him come to the clinic again and repeat the test. In a busy clinic where we have seventy-five or 100 people with fourteen or fifteen refractions it is impossible to go through with the minute details of an examination such as that spoken of by the essayist. We get satisfactory results in our own way. In a busy office it is well to depend on the objective test and expect the patient to accept the glass found with the careful retinoscopy.

DR. POTTER (closing discussion): There are only a few points I care to pay any attention to. They are latent hyperopia, mixed astigmatism, spasm and the testing of children. These are one and the same so far as the method of testing is concerned.

Latent hyperopia is to be developed by slight overcorrection and allowing the patient to use the lenses for close work from ten to thirty minutes when it will be found very often that vision has improved from 20/30 as found by the first part of the test to 20/20 after having worn the lenses.

Spasm is overcome in identically the same way, except that it will probably require a little more time in the fogging part of the test. It is surprising how quickly a muscle spasm will relax under a proper correction for astigmatism and a sufficient amount of spherical correction.

Mixed astigmatism should not give any more trouble than ordinary astigmatism except that it takes a little more time to obtain the exact combination of sphere and cylinder and determine the exact axis.

So far as children are concerned, their testing is a bone of contention. When I first began to work I did not ordinarily attempt to make a fogging test of a child under 12 years of age. Now I am willing to go to the age where they have been in school less than a year and are sufficiently intelligent to answer questions and read the letters on the chart. Illiterates and young children must be tested under a mydriatic unless one is to take an unnecessary amount of time.

I admit I put in more time in refracting than many, but the amount of time I personally take in making a test is only about twenty minutes, because the office assistant makes the preliminary part of the test. She also writes the prescription, takes the lenses out of the trial frame and puts the special lenses into the frame for the patient to use while I am caring for the next patient. It is not difficult to train an office assistant of ordinary intelligence to do this work. I have had four high school girls who have learned the work quickly.

ORBITAL CELLULITIS

F. E. WALLACE, M.D.

PUEBLO, COLO.

REPORT OF CASE

Dr. T., who was called during the morning of July 1 to see D. P., gave me the following history:

History.—A boy, aged 12, an Italian farmer lad, healthy and active, two days previous to consultation, had noticed a twitching and drawing sensation in the tissues of the nose, which had developed by evening into a slight pain at the base of nose and in the right frontal region. A slight swelling of the right upper eyelid was then seen. Redness of the conjunctivae was noticed the following morning, with a greater degree of swelling of upper lid and an extension to the lower lid with slight pain in eyeball, orbit and forehead. The symptoms had rapidly increased by the following day when the physician was called.

Examination.—He found the right eyelids very edematous with swelling and induration over temporal muscle and with considerable redness of the lid tissues and conjunctivae. Epiphora was slight. The lids were nearly closed. The lad complained of pain in the eye and the frontal and temporal region, also of slight headache. The temperature was 102.5 F., the pulse 90. Ice applications were advised and small doses of calomel prescribed. The physician was called at midnight of the same day because the symptoms were becoming more severe and delirium was developing. The temperature was 103.5, the pulse 100. The patient was removed to a hospital and admitted at 2 a. m., July 2. The temperature was 103.5 in the axilla, the pulse 100. By 6 a. m., the patient was more delirious. The temperature was 104 in the axilla, the pulse 120. The swelling of temporal region had slightly receded, because of the ice. The nurse recorded that the patient had been very restless and had had a miserable night.

I saw the patient at 8 a. m. All the symptoms had increased. I found the right eyelids closed with marked edema of both lids. The lids could not be opened by spontaneous efforts. The skin around the orbit was swollen, the temporal muscle indurated, and the eye very prominent. The skin of the lids was purplish red and boggy, but showed no evidence of erysipelas. The conjunctivae of the eyeballs showed marked hyperemia and were so chemotic that a roll was formed around the cornea. This roll was so edematous and boggy that the cornea showed but half its normal diameter. The secretion was of a thin mucopurulent character and moderately profuse. The cornea was not affected and the pupil reacted normally. There was no dilatation. The

vision was lowered to counting figures, the eyeball was fixed, and diplopia was present. Pain was complained of, as was a sense of pressure. The tension of the eyeball was raised. The patient had a fear of impending death. He was very talkative. His attention could be obtained to answer questions. The fever ranged as high as 104 in the axilla, the pulse 120. Vomiting did not occur.

The history and symptoms of nasal, throat or ear infection were negative, as was that of injury or the presence of contagious or infectious diseases. The teeth were all perfect. I could find no evidence of metastasis. The urinalysis was negative. The ophthalmoscopic findings were limited to a haziness of the vitreous. Etiologic factors were entirely obscure. There was no pointing of the abscess. Indeed, the external tissues were so boggy and edematous that fluctuation seemed present at any spot on either lid.

Operation and Treatment.—I advised operation at once. The advice was accepted. Presuming that a suppuration of the ethmoid might be at the bottom of the trouble, I made an incision through the eyebrow down to the bone, loosened the periosteum and probed backward and inward. I was rewarded by seeing pus in small quantities come forward. A small incision was made into the tissues of the upper lid with negative results. Smear examination of the pus showed the *Micrococcus catarrhalis* predominating with a few pneumococci and staphylococci present. A culture also showed the growth of all these organisms. Codein was ordered, as it was necessary to keep him easy. By 1 p. m. the temperature was 101.25 in the axilla. The temperature up to July 4 continued from 101.25 to 104 in the axilla. But little change was noted in the other symptoms. On this date, pus was pointed and was self-punctured at a point beneath the lower eyelid to the outer side. The patient showed great restlessness. From July 4 to 6, the temperature ranged around 102. It was noted that the tissues over the temporal region had become boggy. A puncture 2 inches above and anterior to the ear obtained pus. July 6, the left eye now showed signs of irritation. The lids were becoming edematous. At 5:30 p. m., July 6, his physician injected 10 minims of mixed infection phylacogen.

Clinical Course.—July 7, 8 p. m., the left eyelid was very much swollen. The conjunctivae were hyperemic and chemotic. The temperature was 101 in the axilla. By evening, the symptoms of the left eye were very much worse. The exophthalmos was slight and the eye immobile. The general condition and that of the right side remained unchanged. Pus was coming from the three openings.

At 5 p. m., 20 minims of phylacogen. July 8, 8 a. m., the temperature was 98.6 in the axilla. The edematous conditions of both sides were somewhat reduced. At 6 p. m., the temperature was 101 in the axilla. Twenty-five minims of phylacogen were injected.

July 9, 8 a. m., conditions improved a trifle. The temperature was 100 in the axilla. Delirium was still present although less marked, and there was less restlessness. It was noted that some incoordination of lower limbs was present, while the patient was lying down, or standing, or attempting to take steps.

July 10, 8 a. m., the temperature was 99.8 by mouth, all symptoms improving. Twenty-five minims of phylacogen were injected. The edema was receding. A slight movement of both eyeballs was noted.

July 11, the patient was more rational. The temperature was 100 by mouth. There was less restlessness. The discharge continued from all openings but was lessening. Coordination of the limbs was reestablished. The left eyelid was less swollen and the eye movements were nearly normal. The appetite was improving.

July 12 to 20, the temperature ranged from 99 to 100. July 14, there was general improvement, and the delirium was gone. Movements left eye were normal and movements of right were nearly so. The patient was removed to his home. The operation wound in the eyebrow was allowed to close. Some pus was still coming from the lower opening and the temporal wound. At intervals of two days, small doses of phylacogen were administered by his physician. I saw the patient again, July 22. I found that a small symblepharon had formed at the point of self-puncture and that a trifle of pus was draining there. The scalp wound was draining freely. The prominence of the eye and the edema of lid and of the conjunctivae were very much reduced. The vision was normal. A gradual improvement continued until July 26, when the temperature shot up to 102. The scalp was found to be boggy at a point 4 inches above the right ear. The patient's physician made an incision and evacuated considerable pus. His temperature, however, remained up until July 30, when it became normal.

On this date, I again saw the patient. Both scalp wounds were draining freely. The right eye was still a trifle prominent and the lid was slightly swollen. Pus was coming from the lower lid opening in very small amounts. Right eye movements were not quite back to normal. Injury to the optic nerve was not discovered, neither did a thrombosis of the vessels or detachment of retina occur.

Results.—No opportunity had yet been presented to have a roentgenogram taken, nor had we thought it at all probable that a Wassermann examination would shed any light on the case. I acknowledge that a very painstaking and thorough investigation of the sinuses must be undertaken, in order that the etiologic factors may, if possible, be determined and that, if diseased sinuses are found, they should be thoroughly cleaned, so that a recurrence may not take place. The patient's condition and lack of proper facilities at his home has thus far prevented this investigation.

Word from his physician just before I left for this meeting said that the temperature was practically normal at all times, pus from self-puncture was nil, and the patient was in a favorable condition, although he had a slight discharge from each of the scalp wounds.

ETIOLOGY

Although comparatively not a rare condition, orbital cellulitis is of so serious and distressing a nature that the report of an additional case cannot but be of interest and, I trust, of some value. Cases involving one eye alone are probably not rare, but, from my own experiences and reading, a case in which both orbits are involved would seem to be one of the extreme rarities.

That we have involvement of the orbit from diseases of the nose and accessory sinuses is acknowledged without argument. To establish this relationship is not always possible. It is sufficient to say that disease of the orbit is excited through disease of the nasal cavities by direct communication, because of anatomic arrangement, through toxic absorption, or possibly, as suggested by Parker, through nervous reflex action. He says, "It is not always easy from subjective symptoms alone to determine whether the origin of the syndrome is in the eye, the nose, or the sinus. The fact that an eye lesion coexists with a sinus infection is not in itself evidence enough to establish a causal relation."

I take it that authorities agree that any of the pus-producing micro-organisms may be responsible in causing cellulitis. However, cellulitis may be of nonsuppurating variety. In the non-suppurating type, exophthalmos may occur temporarily, and is due to an inflammation and edema. It has been stated by some writers that the disease is rare in children of less than 9 years, because of the undeveloped conditions of their accessory sinuses. However, numerous cases are more recently reported. Wood writes of a case reported by Hansell. The case was that of an Italian baby of 4 months, who developed an orbital cellulitis without known cause, which terminated in death from general erysipelas. The necropsy showed the accessory nasal cavities to be normal. Bryan recently reports a case occurring in a child 18 months old, from diseased ethmoids.

CONCLUSIONS

We may come to the following conclusions:

1. The etiologic factors are so numerous that a cellulitis of the orbit may arise from any inflammatory condition of any of the proximal tissues.

2. Not infrequently, it may arise from diseased parts far distant, namely, the pharynx, mastoid, tonsils, alveolar processes, suppurative parotitis, the teeth, or indeed from a thrombosed cavernous sinus.

3. The wall between the orbit and the ethmoid cavities being so thin, there can be no doubt that because of disease of the latter cavity, we have extension to the orbit more frequently than through any of the other accessory sinuses.

4. Investigation would seem to show that any of the pus-producing micro-organisms may be implicated.

5. Because the majority of cases arise from diseased ethmoid sinuses, we may get our first indications of pus to the inner side of the eyeball.

6. Cellulitis may become so acutely general that one cannot say at what point the pus may make exit.

7. If the etiology is unknown and the fellow eye is involved, and returns to normal conditions by resolution, we probably cannot determine whether the symptoms were those by direct communication or from toxic absorption.

8. Children may have well developed sinuses.

9. Death from orbital cellulitis is not a rare occurrence.

Points of interest in the case history are:

1. Acute onset.
2. Early formation of pus.
3. Cerebral symptoms.
4. Continued high temperature.
5. Great restlessness.
6. Great edema.
7. Pronounced exophthalmos.
8. High tension.
9. Partial impairment of vision of right eye.
10. Adhesions of lower lid of right eye.
11. Involvement of right temporal muscle.
12. Evacuation of pus by three operative points.
13. Self-evacuation at one point.
14. Involvement of fellow eye.
15. Immobilization of both eyeballs.
16. Left eye returned to normal by resolution.
17. Incoordination of lower limbs for two days.
18. Return to normal vision of right eye.
19. Lack of injury to fundus of the eye.
20. Etiology unknown.
21. *Micrococcus catarrhalis* predominating in pus evacuated.

DISCUSSION

DR. W. W. PEARSON, Des Moines, Iowa: I am sure we have all enjoyed the recital of this case history. The title, I believe, was "Orbital Cellulitis." It is a question in my mind how much orbital cellulitis was present in this case. It reminds me a little of an extradural abscess, not an abscess of the brain tissue. Did he not have simply an ethmoiditis that discharged at the outer angle of the eye? Its course was subperiosteal and necessitated opening and drainage of the temporal region, and extending across the face the infection involved the other eye. Was it not simply subperiosteal extension?

I saw one or two very interesting cases in the past year. The first was that of an officer at Camp Dodge before adequate camp facilities were developed to care for cases of this kind. He was a Yale graduate and an athlete in college. He had a severe nasal infection, a complicating ethmoiditis on one side and a high temperature. Just at the time he was brought to the office Colonel Todd happened to be there, and also Major Black. I asked them to see the case and had a roentgenogram taken, an aid which Dr. Wallace did not have. The plate demonstrated an opacity in one of the anterior ethmoid cells. Pus was draining from under the middle turbinate and I advised drainage. This I did the following morning through the upper inner angle of the orbit, being careful to follow down between the periosteum and the bone. Septicemia developed and he was confined to the hospital three months. He developed a metastasis of the pelvis and it was necessary for the general surgeon to interfere. He afterward went under private care in his Northern home city and I think it became necessary to drain further some pelvic foci. There was also a more or less persistent nasal condition. That man did not have orbital cellulitis, but he did have pressure of the orbit.

The same condition happened in a child of 9 last year, with a much more gratifying result. Simply opening the orbital periosteum and slipping down between the periosteum and the ethmoid cells permitted the escape of considerable pus. A drain was inserted, and in a matter of ten days it healed. There was orbital pressure with a proptosis which remained some time; I felt that possibly it had been permitted to close too early and the line of drainage should have been reopened. Those who consulted with me took a different view and nothing more was done in the case; it has cleared up since and there has been no recurrence.

I think Dr. Wallace was handicapped by not having a roentgenogram, by not draining earlier and so preventing the extension. I take it, the symptoms of incoordination resulted from the toxic condition causing the high temperature and not from bacterial involvement of the cerebral apparatus. I can understand how one not having a roentgenogram must follow the line the doctor did.

I recall recently seeing another case, sent by an oculist from a nearby town. I do not recall all of the symptoms at this time, but greatly to my surprise he removed the eye. This subperiosteal condition I have many times seen get well simply by the employment of proper drainage. I recall another case of several years ago where I had gone subperiosteally almost to the apex of the orbit. That young man came from a neighboring town with a history of proptosis for five days; the vision of the eye was ability to count fingers at 1 foot; there was no temperature and the pulse was normal. There was no history of accessory sinus infection. I opened subperiosteally and was rewarded by finding the pus. Fortunately the optic nerve had not been pressed on

too long to interfere with the recovery of vision. I am not sure the color sense was right even after a year.

In true orbital cellulitis we have interference with the venous return, all degrees of tissue necrosis and advancing sepsis. My observation in erysipelas cases where we have drained the orbit is that the general systemic condition has been so acute that practically all have died. I have seen one or two cases of true orbital cellulitis following cystitis. Of course, in cases of this type, the capsule should be opened and the orbit drained in such a manner as not to interfere with the extrinsic muscles of the eye. Dr. Wallace's case is most instructive and we enjoy hearing a full history of a case of this kind.

DR. JOHN GREEN, JR., St. Louis: Let me relate one case that shows that the clinical symptomatology may sometimes be misleading. A 9-year-old boy was brought into the clinic with a history of having been struck on the right eyelid by a piece of chalk two weeks previously. The lids began to swell and the eye pushed forward. The appearance was typical of acute orbital cellulitis—exophthalmos, lids boardlike, intense pain and fever. Nasal examination was negative. On admission I made several incisions in the orbit fully expecting to find pus, but there was none. I put in a drain, believing that somehow the abscess cavity had escaped me. No pus appeared and the exophthalmos increased. The orbit was then dissected and a small piece of tissue excised, which proved to be sarcoma. Exenteration of the orbit failed to check the process and the child died later from recurrence.

DR. WALLACE (closing discussion): Dr. Pierson asked if we really had an infection of the orbital cavity or not. One can readily understand that pus, having its origin in the ethmoid or sphenoid cells, can burrow between the periosteum and the bone and make exit at any point of the orbital cavity anteriorly. Going back to the history of the case as presented, you will note that I opened up the tissues above and secured pus and that subsequently pus made exit, by self puncture, below and to the outside. It might have been possible, but I do not believe probable, that I was dealing with infection of both sphenoid and ethmoid cells of the same side and that each found exit by a separate route. One above, through the operation wound, and the other below, through the self-punctured opening. Therefore, it is my opinion that I was dealing with an orbital cellulitis *per se*.

Dr. Feingold asks how the symblepharon formed and how cellulitis developed in the opposite eye. You will remember that I stated the symblepharon had formed at the self-punctured opening. It could not self-puncture without pressure and an inflammation of the tissues with subsequent necrosis. This opening was in the culdesac of the lower lid, to the outer side. With the intense inflammation of the tissues at this opening, together with the extreme pressure of the lid against the eyeball, one can understand the breaking down of the conjunctivae of the lid and that covering the eyeball. In the healing which took place, these raw surfaces united to form the symblepharon. Of course this adhesion is not extensive and will not prove detrimental. The most likely explanation as to how a cellulitis developed on the opposite side is because I may have been dealing with a pansinusitis. The nasal history, however, was negative.

You will recall that the inflammation of the left side subsided. This may have been due to a self drainage through the nasal cavity or by absorption. Might it be possible that there was not a true cellulitis? Were we dealing with toxic absorptive symptoms? The symptoms were so severe, however, that I cannot bring myself to believe in the latter explanation.

SOME OBSERVATIONS ON ASTIGMATISM

JOHN GREEN, JR., M.D.

AND

WILLIAM F. HARDY, M.D.

ST. LOUIS

Astigmatism has generally been divided into (1) direct, or astigmatism with the rule (meridian of greatest curvature vertical or nearly so), (2) inverse, or astigmatism against the rule (meridian of greatest curvature horizontal or nearly so), and (3) oblique astigmatism (meridian of greatest curvature in the vicinity of 45 or 135 degrees). Some writers omit the third subdivision and classify these oblique astigmatisms with the first group. As we¹ have recently pointed out this classification is purely arbitrary, as these cases might with equal reason be classified with astigmatism against the rule.

It is singular that an exact terminology has not been adopted for this most exact subdivision of ophthalmic science. One has only to peruse the chapters on refraction in our textbooks to learn that each writer has felt at liberty to make his own definitions which are generally characterized by a vagueness incompatible with scientific precision. We note that even Dr. Jackson,² whose careful study of changes in astigmatism is so illuminating, speaks of direct and inverse astigmatism with meridians of greatest curvature "approximately" vertical and horizontal.

In order to eliminate any uncertainty in the future as to what the boundary lines of the three groups are, we propose the following definitions:

ASTIGMATISM DEFINED

Astigmatism with the rule, or direct astigmatism, shall include all cases with meridian of greatest curvature at any point on the arc from 46 to 134 degrees.

Astigmatism against the rule, or inverse astigmatism, shall include all cases with meridian of greatest curvature at any point on the arc from 0 to 44 degrees and from 136 to 180 degrees.

1. Green, John, Jr., and Hardy, William F.: *Astigmatism Against the Rule*, Precession Pamphlet, Section on Ophthalmology, A. M. A., p. 96, 1918.

2. Jackson, Edward: *Common Changes in Regular Astigmatism and their Causes*, Precession Pamphlet, Section on Ophthalmology, A. M. A., 1918, p. 118.

Oblique astigmatism shall include all cases of astigmatism with meridian of greatest curvature exactly at 135 degrees and at 45 degrees.

It has been argued that there is no necessity for any subdivision, for the reason that practically every eye possessing an astigmatic error should wear a cylindric correction and that it is immaterial whether the meridian of greatest curvature is in the vicinity of 90 or 180 degrees. Theoretically, this contention has merit. But we are convinced that it is useful, on clinical grounds alone, to differentiate at least between direct and inverse astigmatism for the reason that the latter presents certain features of special interest and importance. Our study indicates that inverse astigmatism is more frequent than is generally surmised (in 1,024 eyes with regular astigmatism we found 283, or 27.7 per cent., inverse). Furthermore, as Theobald³ has pointed out, an eye with this type of error is much more capable of "hiding it away" than is an eye with direct astigmatism. This observation accords with our own experience. We are convinced that prior to our systematic use of Lancaster's astigmatic charts, many examples of low inverse astigmatism eluded us. In our series, too, we determined by neutralization of spectacles prescribed by ophthalmologists who had preceded us, that more than half of the men had failed to incorporate a cylinder to correct an inverse astigmatism. Glasses prescribed by refracting opticians only very rarely indicated any appreciation of the existence of this type of error. Low inverse astigmatism gives rise to especially annoying symptoms, such as headaches, blepharitis, congestion of the globe, etc. It is less well borne by the eye than an equal degree of perpendicular astigmatism.

One of the principal reasons for the greater degree of asthenopia in eyes with inverse astigmatism is the greater interference with the legibility of ordinary printed type. In direct astigmatism the vertical components of letters are distinct, whereas the opposite is the case in inverse astigmatism. Letters with vertical components distinct are more easily recognized than those with horizontal components distinct. One has only to render one's self artificially astigmatic with the rule and then artificially astigmatic against the rule (using the same cylinder) to appreciate the difference in distinctness of the same line of letters. This difference can also be beautifully demon-

3. Theobald: Is Astigmatism a Factor in the Causation of Glaucoma? *Trans. Am. Ophthal. Soc.*, 1888, No. 24, 126.

strated by the camera. In the accompanying illustration the upper group of letters was photographed with an ordinary lens, the second group with a lens rendered astigmatic with the rule and the third group with a lens rendered astigmatic against the rule (using the same cylinder). No one will fail to appreciate the lessened legibility of the third group.



Showing the difference in distinctness of letters when a normal lens and astigmatic lenses (both with and against the rule) are used in photography.

It is probably an accurate statement of fact that one finds frequently that for which one is on the lookout. This is exemplified in the search for astigmatism and particularly for inverse astigmatism. That we all possess some amount of astigmatism is a truism; whether it be corneal or lenticular is beside the point. Having detected a small amount of astigmatism against the rule, it does not follow that it should be corrected in all instances. In the treatment of this condition the exercise of judgment is as necessary as in all refractive work.

TWO REQUIREMENTS IN REFRACTIVE WORK

In the correction of refractive errors there are two things to be subserved, namely, improvement of vision, and the increased comfort of the patient. If clarity of vision is gained without comfort, or comfort attained with no improvement in vision, the result has fallen short of the ideal. The average patient will greatly prefer a correction which yields fairly good vision with perfect comfort to that which gives the most acute vision with an aggravation of his discomfort. And occasionally this is just the effect of incorporating a cylinder in the correction. We feel, therefore, that for some patients, especially elderly ones, who for years have been completely comfortable with spheres, it is ill advised to prescribe an astigmatic correction which, theoretically, is indicated. The confusion and discomfort incident to the new order of things offset any increase in visual acuity. Many of us are creatures of habits and even of bad habits.

THE USE OF CYCLOPLEGICS

On the other hand, when it is definitely demonstrated that both vision and comfort are increased by an astigmatic correction, it is incumbent on us to determine with all possible accuracy the strength and axis of the cylinder. And this may necessitate the use of cycloplegics in certain individuals above the age when such drugs are usually used. We believe this to be good practice. We do not believe that a drug like homatropin ever produces glaucoma. It may precipitate an attack, but does not cause the disease. It goes without saying that a careful examination of the fundus, tension and field should precede the use of the cycloplegic in these cases. Hence the presbyopic patient need not be deprived of the advantages of a test under cycloplegia, when the precycloplegic test has given unsatisfactory or conflicting results. It might appear superfluous to labor this point were it not for the fact that a large number of prominent oculists are unalterably opposed to the use of cycloplegics in adults of whatever age.

The advice of Clarke⁴ that the full amount of astigmatism should be corrected cannot be followed in all cases with comfort to the patient. In numerous instances the postcycloplegic test reveals the fact that the patient is either made uncomfortable with the full cylinder or that the visual acuity is reduced. In either event a glass of less cylindric strength must be given.

4. Clarke: *Brit. Jour. Ophthalm.*, 1918, 2, 323.

For instance, a plus 1.5 cyl. of a total of 2.5 may be all a patient will accept at the first test. Some time later plus 2 is accepted, and finally the full cylindric correction is worn with comfort. It is not to be inferred that the astigmatism in such cases has changed. These facts have recently been emphasized by Colonel Elliot.⁵

OVERCORRECTION TO BE AVOIDED

One must be careful not to overcorrect an astigmatic error even in the slightest degree. It is better to err in the opposite direction. Whipper⁶ has recently stated that a patient should be left with a quarter diopter of astigmatism with the rule uncorrected, as this is the normal condition and even essential for comfortable, if not perfect, vision.

MIXED ASTIGMATISM

Always a difficult problem is the management of cases of mixed astigmatism in which there is accommodative cramp. It is very disconcerting to prescribe a glass on the basis of a cycloplegic measurement only to find that the patient complains bitterly of poor distant vision. Various measures such as prolonged atropinization, rest of the eyes, change of occupation, sedatives, etc., often do not suffice to overcome the exalted tone of the ciliary muscle. It has seemed wise in some cases of this kind to yield to the demands of the patient for good distance acuity by increasing the strength of the minus spherical element. We have not had cause to regret this departure from scientific accuracy.

Not rarely do we encounter cases in which one eye shows a low regular astigmatism, whereas the other eye presents a high degree. The cause for this discrepancy is often obscure. Without doubt a number of unilateral cases of high astigmatism are the result of trauma occurring at birth. The following is an illustrative case:

REPORT OF CASE

In 1912, baby, L. O., was seen by one of us four days after birth. The obstetrician stated that one blade of the forceps had pressed firmly over the child's left eye. The cornea was entirely opaque, but there were no inflammatory signs. Under dionin and hot applications, the cornea cleared. A few days ago this little girl, now 6 years old, was brought to us because the mother noticed that the child in looking at picture books had a tendency to close the left eye. Right vision = 6/5; left vision = 6/40.

5. Elliot, Colonel: *Brit. Jour. Ophthal.*, 1918, 2, 313.

6. Whipper: *Ophthal. Rec.*, Vol. 24, 296.

The ophthalmometer showed R, As. 1 mc, 90; L. As. 6 mc, 90. Both corneas were perfectly regular. Oblique illumination showed the presence of three vertical striae situated in the deeper layers of the cornea, one coinciding with the inner pupillary margin, the second with the other pupillary margin, the third midway between the latter and the outer limbus. A skiascopic examination disclosed a regular astigmatism plus 4.5 D. with meridian of greatest curvature at 90 degrees. With plus 4 cyl. axis 90, L. V. 6/10.

IRREGULAR ASTIGMATISM

Every one possesses a small amount of irregular astigmatism which is, of course, not perfectly correctable by any cylindric lens. Whenever there is a deformation of the cornea from injury or inflammation or when the lamellae of the lens are irregularly disposed or swollen as in incipient cataract, there is bound to be more or less irregular astigmatism which may or may not be associated with the regular type.

In the presence of irregular astigmatism, whether of corneal or lenticular origin, it is thoroughly worth while to determine with the greatest care the cylindric or spherocylindric combination which yields the highest vision. When the irregular astigmatism is a more or less fixed quantity, as for example, in an eye with a cornea deformed by a long past keratitis or injury, a painstaking correction with spherocylinders will often be highly acceptable to the patient who is grateful for the heightened acuity thus secured. But every case is a law unto itself; not rarely a simple sphere, which yields less vision, is preferred to any combination.

An eye with an incipient cataract which has previously rejected all cylinders, may often be made to see very well with a spherocylindric combination. But here again it is often a case of "Love's Labor Lost"—the combination is soon found uncomfortable or visually unsatisfactory. We presume that the explanation lies in the fact that the irregular lenticular astigmatism is constantly changing with the progress of opacification.

DISCUSSION

DR. WILLIAM H. CRISP, Denver: I believe that the oldest patient in whom I have used cycloplegia was between 50 and 55 years of age. In this particular case I was able to reveal more hyperopia than I could uncover by the fogging method, and the patient wore the correction with comfort. In the case of one or two presbyopes, long trial of the correction given under a cycloplegic did not result in complete relaxation of the accommodation.

Speaking generally, my preference is to insist that every patient shall give a prolonged trial to the full correction of hyperopia, rather than to

give the patient a lower correction first and tell him that it may need to be increased. I am convinced that as a general rule, if the confidence of the patient is obtained, the giving of the full correction from the start is the more satisfactory method. I have had a large number of cases in which the patients were very much disgusted with their glasses at first, but subsequently became very comfortable. One woman, a bank cashier, wrote after several months thanking me for having insisted on her perseverance.

It is necessary, of course, for every one of us to realize that now and then our very best efforts do not yield an absolutely final measurement of the patient's refraction. This may possibly be due to there having been a good deal of previous strain, with perhaps some irregular contraction of the ciliary muscle, producing a slight difference between our original correction and the patient's actual requirement. If I suspect that this may occur I tell the patient that we may have to make a slight change in a month or two. The thing is to get the full cooperation of the patient.

As regards the classification of astigmatism into with the rule, against the rule, and oblique, I do not care much what you call the particular arrangement of a patient's astigmatism. The essential is to measure accurately the strength and the axis in the individual case. I have had a great deal of satisfaction from the use of the combination of the astigmatic dial with the cross cylinder along the lines which I suggested a year or so ago (*Annals of Ophthalmology*, January, 1917) using very largely the so-called Thomas astigmatic chart consisting of a cross which can be rotated into any position. By rotating the cross cylinder while the patient looks at this chart, and asking the patient to indicate with which position of the cross cylinder the two lines of the cross look more nearly alike, one may often obtain a greater refinement as to the strength of cylinders required than by using the chart without the cross cylinder; and at the same time the estimation is often more reliable than by using the cross cylinder with the test type.

DR. H. N. BLUM, New Orleans: Referring to astigmatism against the rule, in our clinic in New Orleans 50 per cent. of our patients are negroes, and I have found that about 35 per cent. of the errors of refraction in the negro have been astigmatism against the rule. I do not know why, but it is a fact.

I think we should be tolerant of our predecessor in the matter of neutralizing lenses, since the glasses worn by the patient are not a fair test of the other man's work. I find it difficult to get patients to come back once, much less the second or third time, in order to have their glasses examined.

DR. EDWARD JACKSON, Denver: Classification is a matter of convenience, and it is a rather important matter to understand what we are saying to each other to have a universally recognized term for each form. What is direct astigmatism, and what is inverse? If we confine these terms strictly to the greatest curvature at 90 degrees for direct, and inverse for the greatest curvature at 180 degrees, they apply to a quite limited number of cases that can be indicated by 90 degrees and 180 degrees without another term. The thing is to have understood as direct astigmatism a certain considerable class, in which the meridian of greatest curvature approaches 90 degrees. The probability is these are similar to some extent in the deformity of the eyeball, and the inverse are alike to some extent. To divide the 360 degrees, or 180 degrees, into three regions, giving each a distinctive name, would serve all purposes for discussion. Direct astigmatism at approximately 90 degrees, or go either side for 30 degrees; inverse at 180 degrees, and go either side 30 degrees,

and the oblique coming between these two regions. The careful statistical studies already made all indicate that astigmatism against the rule, so-called, is much more common than formerly supposed, and I think there is a distinct move in that direction with increasing age of the patient. This should be considered in that connection.

DR. L. H. TAYLOR, Wilkes-Barre, Pa.: I presume few present are old enough to remember a paper read by Dr. Chisholm, entitled "The $\frac{1}{4}$ D. Lens; the Most Important Lens in the Case." He dwelt on that at length. I think we were inclined to doubt a little, but attention was called to correcting the low grades of astigmatism, particularly those against the rule. I recall a case of a practitioner of medicine several years ago, with headaches and difficulty that made him think it absolutely necessary to give up his practice. I thought the matter very serious, but suggested the correction so far as his eyes were concerned; but we found only a low grade of astigmatism. But by correcting that he was able to go on practicing medicine ever since, and he has a large practice. Another thing, in regard to having the patients bring back the glasses for tests. There are advantages and disadvantages. Those of us living in towns where we see these people and order glasses for them have the advantage of seeing and testing the glasses, and I never allow patients to receive their glasses without testing them to see that they are absolutely correct. Sometimes I find a slight error. We should not allow the patient to take the glasses from the optician without having an opportunity to test them and see that they are correct.

DR. GREEN (closing discussion): Dr. Crisp prefers to give a full correction at once, relying on moral suasion to get the patient to wear this correction. If he has succeeded in doing this he is open to congratulations. I usually begin with the glass which is accepted on post-cycloplegic test, but insist that the patient return at a stated interval, impressing on him that this later visit is an integral part of the examination. I recall Dr. Crisp's paper in which he describes the conjoint use of the cross-cylinder and astigmatic chart. I have long been in the habit of checking up results by having the patient observe the cross arms of the Lancaster chart and then hold a plus 0.25 cylinder with the axis parallel to one arm of the cross and then parallel to the other. Dr. Blum believes that from 30 per cent. to 35 per cent. of negroes have astigmatism against the rule. That percentage does not differ very much from the percentage in our series (27.7 per cent.). Our patients were all white.

The classification of astigmatism to which Dr. Jackson has alluded seems to us fairly important. Our definitions may or may not be accepted, but we have felt the need of replacing indefiniteness by definiteness.

Dr. Jackson's point that astigmatism against the rule increases with age is well taken. Nevertheless, in our series we found that nearly as many examples of astigmatism against the rule were below 40 as above.

ETIOLOGY OF EYE DISEASES

FREDERICK F. TEAL, M.D.
LINCOLN, NEB.

A review of my cases, marked either by a failure to cure or by an exasperatingly long time in obtaining favorable results, leads me to a short consideration of the etiology of eye diseases. The same trend of thought might well apply to any other group of diseases, and a study of the subject certainly proves interesting, if not as satisfactory as one might wish.

Etiology is so intimately related to treatment in the daily round of the oculist's work, that time spent in the pursuit of a morbid cause is necessarily profitable. Too much credit cannot be given those indefatigable clinical and laboratory workers of past and present times, whose labors have made it easier for us to combat the enemy, disease. Time does not permit me to name them nor to enumerate the results of their investigations. But for those who are ambitious to distinguish themselves in ophthalmology, there is still a vast territory in which to work; and what a satisfaction to the conscientious investigator to uncover a vein of fact that points to a new discovery, be it great or small.

It is with this thought in mind that I attempt to point out one or two paths that we might tread with profit to ourselves and to others.

IRITIS—GENERAL CONSIDERATIONS

Let us for a moment consider iritis. For years the prevention of its recurrence has baffled our efforts. By persistent effort in the study of its etiologic data, the profession now has the expectation of a fairly hopeful prognosis, and yet the action of this association in appointing a committee to thoroughly and scientifically investigate the etiology of iritis is proof enough that the question is not yet settled.

In some ways our progress of ocular etiology as a study has been slow, and, as I see it, after a hasty review of a few of the textbooks printed in the last forty or fifty years, it is partly because we embrace a new idea with too much alacrity, work it to death, and then perhaps reverse ourselves when some new idea is accepted. This results in our sitting back and resting

complacently while the new fad is on, and later, when it is abandoned, a proposition that perhaps really has merit is cast aside.

In De Wecker's "Ocular Therapeutics," published in 1879, we find iritis classed as syphilitic (or gummatous), rheumatic (or gonorrheal), arthritic, and blennorrhagic. To show the rather chaotic condition of the etiologic status of the time, I quote the last paragraph of his chapter on iritis. After classing one form as gummatous or syphilitic iritis he says "Syphilis is the cause of from 60 to 70 per cent. of all cases of iritis, *although the gumma form enters but slightly into these calculations.*"

Meyer in "Disease of the Eye," published in 1887, gives syphilis as the cause of iritis in 75 per cent., the remainder due to rheumatic diathesis and other causes. Noyes in "Diseases of the Eye," 1890, gives syphilis in 50 per cent., gonorrhea, malaria, scrofula, and tuberculosis. His outline of treatment is splendidly given, including measures to combat the etiologic factors.

Norris and Oliver pointed out that, in addition to syphilis, gonorrhea, tubercle and other constitutional factors, those ailments capable of giving rise to inflammations of the iris are intimately associated with, if not actually caused by, micro-organisms. Fox gives, in 1910, the usual list of constitutional causes.

Duane in his translation of Fuchs' last work gives the old list of dyscrasias as a cause, but adds an important thought which I wish to point out and which indeed emphasizes the idea prompting me to write this paper. He queries: "Shall we call every iritis which has no characteristic marks, syphilitic, simply because it occurs in a syphilitic person? A syphilitic person may also acquire an iritis from any cause whatever." The translator also adds, the disease may be kept up, or relapses caused by other infections (for example, oral infections—as emphasized by De Schweinitz). The older members may recall that Dr. Young of Burlington pointed out in years past the idea of autointoxication as a cause.

This warning against the strictly specific treatment of iritis Duane gives in mentioning the rheumatic form, in the following words: "This was formerly regarded as one of the most common causes of iritis. Now the increasingly prevalent view is that it is not a morbid entity at all." In other words, the morbid infections giving rise to the rheumatism, and located in the teeth, tonsils, accessory sinuses, intestinal tract, urethra, and similar structures, is probably the cause of the iritis. The idea

of specific treatment alone has stayed with us a long time but we are progressing.

EPISCLERITIS AND SCLERITIS

If we are fairly satisfied with our knowledge of the etiology and treatment of iritis, suppose we consider for a moment the subject of episcleritis and scleritis. A perusal of the same authors quoted gives rheumatism, syphilis and gout as causes. Fuchs in his last work shows real progress over the older textbooks. He discusses etiology in two and one-half lines and says "Treatment avails but little against it." The custom has been if there was a positive Wassermann reaction to give the specific medicine, and the same was true if rheumatism was present. Let us not fail to recognize that although a scleritis is found in a syphilitic the real cause may be and probably is in some neighboring tissue or organ, and it is our task to ferret it out.

W. Gilbert in the *Archives* for September, 1915, gives a careful pathologic study of scleritis occurring in a patient that he concludes is undoubtedly due to gout, basing his conclusions on the gouty pathology found in the eye. A similar article by Verhoeff states that his case gave a positive Wassermann reaction and he believes syphilis to be the probable cause. Undoubtedly the writers treated these cases on the basis of gout and syphilis, but there is nothing in either article to prove that there may not have been some factor other than the presence of constitutional dyscrasias. Is the etiologic history of iritis to be repeated in scleritis?

Contrast the results of these investigations with a study by Bell of New York. He presented a case of bilateral papilledema before the New York Ophthalmological Society. Some of the members gave brain tumor, others on account of the urinary findings, Bright's disease as a cause. Trephining had been strongly recommended by competent men.

But Dr. Bell is a real clinician, and in his quest for more light found the patient had a chronic ethmoiditis and sphenoiditis. He reasoned that a severe toxin was circulating in the blood. Thorough opening up of the sinuses cleared up the case of papilledema—as well as the kidney lesion.

COMMENT

I have said enough to emphasize the following points:

(a) That constitutional dyscrasias, while important in the therapeutic conduct of our cases, are many times secondary as

etiologic factors and their discovery may act unconsciously as a deterrent in our active pursuit of other causes of the disease under investigation.

(b) That we are still following the beaten path in an etiologic way, when we might be developing knowledge if we would but arouse ourselves to the importance of constant effort in the hunt for the morbid agent of each case as it comes to us.

The laboratory has been of inestimable value in pointing the way to the treatment of disease, and is necessary in our everyday work. The great help given us by the laboratory worker has no doubt had its influence in helping us to forget that the clinician also has his sphere.

Let us not neglect the clinical signs and clues that lie before us, if we but seek for them, and when found, with the aid of the chemist, bacteriologist and pathologist, combine results, to the end that etiology may mean something as a guide to cure.

If 50 per cent. or any other per cent. of our cases of iritis were due to syphilis, then a thorough course of antisyphilitic treatment should have prevented its recurrence. The fact that such treatment has not prevented the recurrence should prompt us to admit it, and seek other causes. Textbook authors particularly should be careful not to mislead the reader, especially the younger one, by too broad statements in discussing etiology.

In this way beginners in ophthalmology will not be guided wrongly and so lead to a lack of faith in therapeutics; teach the younger man that the successful physician combines the indefatigable industry and superior technical knowledge of the laboratory worker with the clear-thinking brain and investigating instinct of the clinician.

DISCUSSION

DR. H. B. YOUNG, Burlington, Iowa: It seems a rather remarkable coincidence that on this, the twentieth anniversary of my first pronouncement on this topic (for it is just twenty years ago that here, in Denver, also with Dr. Gifford in the Chair, I delivered under the name of "Auto-Intoxication Amblyopia," the puling infant now grown apace) I should be asked to open this discussion. It may be remembered that my original paper in the ophthalmic section of the American Medical Association (1898) was a series of case reports with deductions. The first case was the most striking: A woman convalescent from abortion, totally blind for seventy-two hours, restored to normal vision on an exclusive diet of calomel, castor oil and water. As another coincidence I may add in this connection that just eight years later this woman's daughter, two weeks after normal accouchement, was similarly blind, with widely dilated nonresponsive pupils, for thirty-six hours, and made full recovery under the same treatment.

About this time last year, however, I saw a case that in the light of the handling and result is fully as worth recording: A woman, aged 55, robust physically and mentally, a resident of an Eastern city, visiting in the Middle West, had for a year been the victim of recurrent attacks of uveitis which had impaired vision through deposits of pigment on the anterior, and debris on the posterior capsules of both lenses. Extensive questioning about her own history and that of her family, investigation of her somatic functions, and inspection of her nose, throat and mouth, shed alike no light on the etiology. Of the laboratory tests desired I succeeded in getting only roentgenograms of her jaws. One of these revealed the presence of an unerupted third molar; and this, while requiring some imagination as a focus of infection, I advised to have extracted. After a month's delay, and a visit in the meantime to the Mayo Clinic where the sum of information obtained was that the cataracts (?) were not ready for operation, my advice about the molar tooth was followed. The immediate result on healing was a "feeling of relief," although the eyes had previously been quiet for a month. Shortly after this the patient departed for her home in the East, with a promise to report the more remote outcome. This delayed report has come only within the last month, in a letter, from which I quote: "My vision is still poor, but I know that I am better in this respect than I was. I have not had pain or inflammation since that impacted tooth was extracted; and I cannot be too grateful to you for insisting on having it taken out." So it seems that an impacted molar can be listed among the etiologic factors in eye diseases. What next?

DR. MELVILLE BLACK, Denver: The Metropolitan Building is getting the reputation that if you go there you will probably be passed around to a dozen different doctors before you get out. There is a modicum of truth in this. After we have had dental roentgenograms taken and then have another dentist extract a few teeth, and the internist make an examination, and the pathologist go over the urine, etc., the patient will feel as though he had been passed around considerably. But even then we may not have discovered a positive cause of some of these obscure diseases of the eye. These cases bother us greatly and they do not always stay with us. They are very likely to get discouraged and change to some one else. And then the other fellow goes over all these things, because no one believes any one has done it as thoroughly as he will do it. I have a lady at present with a marginal keratitis. She has had everything done, but her condition is not improved. There is an underlying cause of course. But it is not so easy to find it sometimes as we are led to believe.

DR. HAROLD BAILEY, Springfield, Mo.: This is a live subject, and the essayist in his paper touched on some important points that are especially pertinent at this time. The more we study focal infections the more we realize their possibilities for evil, depending in a measure on the nature and kind of the infection and on the power of resistance in the individual. The focus of infection may be around the teeth, in the sinuses, in the tonsils, in the gastro-intestinal tract or in some other part of the body. We know that the bacteria causing the infections produce proteins, some of which are toxic. This degree of toxicity varies according to the kind of the infecting organism, according to the health of the individual and his susceptibility, all of which are factors in the ability of his blood to create those antibodies the quality of which measures his power of resistance. We know that only too often the toxins are given off in such quantities that the antitoxins produced in the blood are inadequate to counteract them and we get an explosion. This explosion may occur in the eye in the form of an iritis or some other manifestation just as it may occur in some other part of the body.

It is also well for us to remember that proteins are taken into the body in the foods ingested. These proteins may not be toxic in ordinary quantities unless the individual is extremely sensitive to certain proteins. In larger amounts, however, as may be the case when an excess of a certain food or foods are ingested by the individual, a protein may be taken in excess and thereby become toxic and also create an explosion or local manifestation in some organ, just as may occur in the case of the toxin proteins produced by bacteria. It may be necessary to vaccinate the individual with extracts from a large number of foods before the reaction is obtained, which shows which article of diet is the real offender. This question of food intoxication is an important field, in which further investigation may throw some light on the etiology of certain eye diseases which at the present seem rather obscure.

DR. JOSEPH S. LICHTENBERG, Kansas City, Mo.: This paper and the discussion has brought out the fact of the interreliance of one branch of medicine on the others, and the determination of the etiology is in group work, and there ought to be groups formed on the idea of Cabot, where these things can be worked out.

ECLIPSE BLINDING

EDWARD JACKSON, M.D.

DENVER

In this connection the term blinding is only appropriate as regards small areas of the retina; and in the majority of cases no permanent impairment of function is produced. The area of retina exposed to the direct light of the sun at any one time in an eye perfectly focused on the sun, is a circle about one-sixth of 1 mm. in diameter, slightly smaller than the fovea centralis. By variable and inaccurate fixation, or by ametropic diffusion, this space may be increased. A circle more than 1 mm. in diameter is involved by diffusion, in myopia of 10 diopters, with a 4 mm. pupil. But such diffusion correspondingly diminishes the intensity of the stimulus, to which any particular part of the retina is exposed.

The pathologic effects of excessive exposure to direct sunlight must have been noticed by the sufferer at all periods of the life of the race. But they could only be studied objectively since the use of the ophthalmoscope. Galen, in somewhat exaggerated terms, stated the dangers of such injuries; and other writers have ascribed the complete blindness of certain persons to this cause. Galileo was supposed to have impaired his sight in this way. But it is doubtful whether there is any more foundation for this belief than the facts that he became blind, and that he had previously made elaborate studies of the sun-spots.

The earliest and least serious symptoms of eclipse blinding, exaggerated after-images, and temporary diminution of visual acuity, have been experienced by nearly every one, from looking at the sun near sunrise or sunset. Astronomers and navigators compelled often to make observations on the sun, have had greater familiarity with the phenomenon; and often have a good practical understanding of the dangers of exposure of the retina to direct sunlight, and the importance of the precautions taken to prevent permanent injury.

EARLY LITERATURE ON THE SUBJECT DEFICIENT

But it is a curious fact that the pathologic consequences of exposure of the retina to the direct sunlight and heat of the sun's rays have, until the last five years, received scant attention in medical literature. Mackay, in 1893, found the best account

of them in the British literature was an abstract by Marcus Gunn of Sulzer's article. There seem to be ten references to publications in medical literature before 1880, but in the index catalogue of the library of the Surgeon-General's Office, up to 1910, only sixteen references were found, under four headings, the earliest going back only to 1885. Since 1911, however, thirty-five such papers have been published, as shown by the *Ophthalmic Year Book*.

In the larger number of cases of eclipse blinding the disturbance is functional, due to excessive stimulation of the retina by light. Or, at most the very pronounced functional disturbances are accompanied only by vascular changes, which may be regarded as an exaggerated physiologic repair. In a second class of cases the functional changes are accompanied by organic alterations, probably dependent on the focussing of heat rays on the retina; and such changes are generally permanent.

When the gaze has been fixed directly on the sun, the affected area is small, there is a single definite scotoma in the field of vision, a greater likelihood of organic change, and the effect is more commonly permanent. If the fixation be shifted during the observation, the resulting scotoma will be larger and may be multiple, the vision for the time being more disturbed; but there is less likelihood of noticeable ophthalmoscopic changes, and a better prospect of complete recovery.

THE ECLIPSE OF 1918

On June 8, 1918, a total eclipse of the sun began in the north Pacific Ocean, entered the United States across the southwest corner of Washington, passed across Oregon, Idaho, the adjoining corners of Utah and Wyoming, Colorado, Kansas, Oklahoma, Arkansas, Mississippi, Alabama, and off the Atlantic coast near Orlando, Fla. It was total over a path about 60 miles wide; and more than half the sun's disk was obscured in every part of the United States.

The danger from such an eclipse arises from the gaze being fixed on it until a pathologic light adaptation occurs in the part of the retina directly exposed to the sun's rays. This makes looking directly at the sun with this part of the retina less unpleasant, and it is continued until the damage is done. The more striking the phenomena of the eclipse, the greater attention it attracts; and the larger the number of people whose interest overcomes their caution. There is an especial danger

when the eclipse is total, from the slender crescent of the sun that follows totality and rapidly increases.

A letter with return postcard was sent to 600 oculists practicing in various parts of the United States, asking them to report the number of cases of damage to vision from watching the eclipse they had either seen or heard about. From these 195 responses were received. The majority of replies indicated that no cases had been seen or heard of. But a minority reported the occurrence of cases of eclipse blinding. The names of those reporting such cases, their places of residence, and the number reported by each are as follows:

CALIFORNIA

| | | |
|----------------------------|--------------------|---------|
| Alexander, E. W..... | San Francisco..... | 1 case |
| Blake, C. R..... | Richmond | 3 cases |
| Cohn, Robert D..... | San Francisco..... | 1 case |
| Dolman, P..... | San Francisco..... | 1 case |
| Glaser, Edward..... | San Francisco..... | 1 case |
| Green, A. S. and L. D..... | San Francisco..... | 1 case |
| Miller, R. W..... | Los Angeles..... | 1 case |
| Thomas, H. G..... | Oakland | 4 cases |
| Sharpe, L. S..... | Los Angeles..... | 1 case |
| Wagner, E. R..... | San Jose..... | 6 cases |
| Walker, A. W..... | Riverside | 2 cases |
| Negative reports | | 15 |

COLORADO

| | | |
|------------------------|--------------|--------|
| Crisp, W. H..... | Denver | 1 case |
| Negative reports | | 8 |

IDAHO

| | | |
|------------------------|-----------------|---------|
| Clothier, J..... | Pocatello | 2 cases |
| Negative reports | | 1 |

KANSAS

| | | |
|------------------------|--------------------|---------|
| Blunk, J. P..... | Ottawa | 1 case |
| Cole, C. W..... | Norton | 2 cases |
| Lidikay, C. J..... | Kansas City..... | 1 case |
| Parker, I. B..... | Hill City..... | 2 cases |
| Rader, J. A..... | Caney | 6 cases |
| Robertson, E. N..... | Concordia | 1 case |
| Smith, T. E..... | Independence | 1 case |
| Settle, J. A..... | Reading | 2 cases |
| Negative reports | | 22 |

LOUISIANA

| | | |
|------------------------|--|---|
| Negative reports | | 1 |
|------------------------|--|---|

MASSACHUSETTS

| | | |
|------------------------|--|---|
| Negative reports | | 1 |
|------------------------|--|---|

MISSOURI

| | | |
|----------------|------------------|---------|
| Curran, E..... | Kansas City..... | 2 cases |
|----------------|------------------|---------|

MONTANA

| | | |
|------------------------|--|---|
| Negative reports | | 1 |
|------------------------|--|---|

NEBRASKA

| | | |
|------------------|------------|---------|
| Benson, H. W. | Oakland | 2 cases |
| Callfas, W. F. | Omaha | 2 cases |
| Gifford, H. | Omaha | 3 cases |
| Golding, D. G. | Fremont | 3 cases |
| Minnich, C. S. | Palmer | 4 cases |
| Shields, W. D. | Holdrege | 4 cases |
| Stevens, Q. H. | Broken Bow | 1 case |
| Zellers, M. T. | Hooper | 2 cases |
| Negative reports | | 6 |

NEW MEXICO

| | | |
|------------------|-------------|---------|
| Clarke, S. C. | Albuquerque | 2 cases |
| Negative reports | | 3 |

NEW YORK

| | | |
|------------------|--|---|
| Negative reports | | 1 |
|------------------|--|---|

OKLAHOMA

| | | |
|------------------|-------|---------|
| Roth, A. W. | Tulsa | 2 cases |
| Negative reports | | 19 |

OREGON

| | | |
|------------------|----------|---------|
| Ainslie, G. | Portland | 1 case |
| Dickson, J. F. | Portland | 1 case |
| Gordon, G. G. | Baker | 2 cases |
| Negative reports | | 9 |

TEXAS

| | | |
|-------------------|---------------|---------|
| Allen, L. | Houston | 2 cases |
| Applewhite, S. C. | San Antonio | 3 cases |
| Hartsook, C. R. | Wichita Falls | 2 cases |
| Hicks, W. D. | San Antonio | 1 case |
| Kopecky, C. L. | Yoakum | 2 cases |
| Ralston, W. | Houston | 3 cases |
| Negative reports | | 45 |

UTAH

| | | |
|------------------|----------------|---------|
| Merrill, H. G. | Provo | 1 case |
| Reynolds, F. O. | Logan | 1 case |
| Stookey, W. M. | Salt Lake City | 2 cases |
| Negative reports | | 2 |

WASHINGTON

| | | |
|-------------------|---------|--------|
| Balabanoff, I. P. | Tacoma | 1 case |
| Seclye, W. K. | Seattle | 1 case |
| Negative reports | | 9 |

WYOMING

| | | |
|------------------|----------|--------|
| Strader, G. L. | Cheyenne | 1 case |
| Negative reports | | 3 |

A total of ninety-seven cases are reported to have followed the eclipse of June 8, 1918. With these were reported four cases of longer standing. In large areas the afternoon was cloudy, so that no one there saw the eclipse. This was true of Northern Texas and a large part of Oklahoma and Colorado. From Texas thirteen cases were reported, all from localities

south of the thirtieth parallel, except two from Wichita Falls. From Oklahoma none were reported; and from Colorado only one was heard of.

In Colorado and in Western Oregon and Washington another factor influenced the number of cases. Through the newspapers a considerable campaign of education as to the dangers and prevention of eclipse blinding had been carried on. This is alluded to by several of those who have sent in reports. The result was that only one case each was reported from Tacoma and Seattle in Washington, and two each from Portland and Baker City in Oregon, and one from Colorado, six in all. In contrast with this, twenty-two cases were reported from a slightly smaller population in California. In the latter state 48 per cent. of those reporting had seen cases; but in Colorado, Oregon and Washington only 20 per cent. had seen cases.

SYMPTOMS

The frequency with which the different symptoms were observed was as follows:

| Subjective Symptoms | |
|---|----|
| Central scotoma or lowered vision..... | 62 |
| Photophobia, asthenopia, lacrimation..... | 7 |
| Dazzling or dancing of object..... | 2 |
| Disturbance of color perception..... | 3 |
| Narrowing of peripheral field..... | 1 |
| Ophthalmoscopic Lesions | |
| Macula dark red, retinal hyperemia..... | 22 |
| Macula gray or edematous..... | 7 |
| Definite white spot near fovea..... | 9 |
| Retinal hemorrhage | 1 |
| Pigment disturbances | 2 |
| Vitreous opacities | 1 |
| External Lesions | |
| Conjunctivitis | 2 |

Central Scotoma.—The reports of these cases were generally extremely meager, some giving no details as to symptoms whatever. Nearly all that mentioned the symptoms complained of included central scotoma or lowered acuteness of central vision. Of 50 eyes without ophthalmoscopic changes, 24 recovered; and in 26 the impairment was likely to be permanent. Of 37 eyes in which ophthalmoscopic changes were observed, 14 recovered, and 25 were permanent. In some there was full recovery in a few hours, in others in a few days. But in some of the cases seen after former eclipses, central scotoma had persisted after many years. The ophthalmoscopic changes followed by recovery

of vision were moderate hyperemia or edema; but in a few there were slight permanent pigment changes. Decided white spots in the retina were followed by permanent visual impairment.

Photophobia, lacrimation and asthenopia were the chief complaint in 7 cases; and in 5 no other symptom was complained of. The photophobia sometimes prevented the observation of other symptoms probably present. This is not the photophobia that is experienced during exposure to the sun. It comes on later, and is probably a symptom of inflammatory reaction in the damaged retina, which occurs only in certain patients. Others presenting retinitis of equal severity may remain free from dread of light.

Dazzling or dancing of light before the eyes was especially complained of in only two cases. But in all probability it was present in many others, and especially in their later stages. Disturbance of color sensation was the chief complaint of three patients; but it occurred to some extent in others. A symptom not mentioned in these reports is metamorphopsia. This generally does not appear until a later period, when vision has partly cleared up so that the distortion of the finer details of objects can be perceived.

Ophthalmoscopic Symptoms.—In more than one-half the cases no ophthalmoscopic examination is reported. The most common change noted in those that were so examined was circulatory. It is spoken of as a dark red color of the macula, or as a hyperemia of the retina. This symptom alone is probably always transient.

An evidence of greater injury to the retina is the gray veiling of the macula by edema, resembling that of commotio retinae. This was observed in seven cases. It also passes off, but may leave prolonged or permanent damage to function.

Definite light or white spots in the region of the fovea were observed in nine cases. Some of the less definite of these lesions may disappear; but the more pronounced of them always leave permanent impairment of vision, a central scotoma, and commonly some metamorphopsia.

The following case reported by Dr. H. Gifford of Omaha is typical:

REPORT OF CASE

A young woman, aged 22, looked at the eclipse without smoked glass and noticed poor sight immediately afterward. She was seen by me June 10, 1918. Best vision with correction was found to be 20/100 each eye. Before the eclipse her vision had been normal so far as she knows. Ophthalmoscope showed in

each eye, dense white sharply circumscribed spot taking in practically the whole fovea. In the right eye this spot is perfectly circular. In the left eye there was a slight projection of the white area at the temporal side. The rest of the fundus was normal. I heard from her again July 1, 1918, and she said that her sight had not been improved.

Anomalous Symptoms.—In two cases marked acute conjunctivitis is reported, as the form of damage caused by watching the eclipse. There seems to be no reason to doubt the probability of such etiology. Prolonged exposure to bright sunlight may cause a kind of sunburn of the conjunctiva. Kerato-conjunctival inflammation is the essential lesion of one form of "snow blindness." It is quite conceivable that the general exposure of the bulbar conjunctiva to the sunlight might be prolonged enough to cause such inflammation, without any single part of the retina having been subjected to the image of the sun long enough to produce a retinal lesion.

In one case *vitreous opacities* were observed. These might have been accidentally coincident. But it was noted that the patient noticed the black specks before the eyes next day after the eclipse; and many small and one large opacity were found in the vitreous a week later $V = 20/30$. After forty days, vision had risen to $20/15$ and but one large vitreous opacity remained.

In one case *narrowing of the general field of vision* was reported. Vision in left eye was reduced immediately after viewing the eclipse without protection. Three days later it was $20/75$ and she complained of a "floating spot, the size of a bean, before the eye." The ophthalmoscope showed the disk slightly pale, and the visual field was markedly contracted. Within two months central vision and the field were nearly normal; but the spot was still complained of. Possibly in this case the central scotoma caused by the eclipse called attention to another condition present which was of different origin.

A condition resembling eclipse blinding, but due to the watching of airplanes in flight, is reported by Dr. Crittenden Joyes of Fort Worth, Texas. He has seen six or seven patients in whom vision was much reduced, temporarily, with central scotoma by looking at airplanes when flying toward the sun. The condition has passed off in a few days. In watching the airplane the image of the sun would not so continuously fall on the same part of the retina as when watching an eclipse; so that the more severe and definite retinal lesions would not arise in this way.

SUMMARY

Eclipse blinding, although long noticed, has been discussed in medical literature chiefly in the last twenty-five years. It is marked by central scotoma and lowered vision, photophobia, asthenopia, lacrimation, dazzling or dancing of the thing looked at, disturbance of color perception and metamorphopsia, noticed subjectively. With the ophthalmoscope the macula often appears a darker red, there is retinal hyperemia, or the macula looks gray edematous, or there may be light spots in the affected area, or a single sharply outlined white spot is seen, or there may be alterations of pigmentation. In rare cases a sharp conjunctivitis is produced.

Following the eclipse of June 8, 1918, ninety-seven cases were reported by 195 observers. Probably ten times this number occurred within the United States. The most of these recovered completely in a few days or weeks; but an important part of them were left with a small permanent central scotoma or metamorphopsia. Much can be done to prevent such injuries by systematic warning of the public of the danger.

DISCUSSION

DR. GEORGE L. STRADER, Cheyenne, Wyo.: I shall not attempt to discuss this interesting paper, except to report a case of my own. A young woman, librarian, aged 25, came to me, June 20, giving a history of having looked at the sun as long as she could see it. Since then her vision has been poor, but was improving. She had at that time 20/50 vision each eye, with very small central scotomas, a slight congestion of the maculae, and in the center of each macula a small glistening white spot, which probably was not caused by the sun's rays. She complained of considerable light irritation. I gave her dark noviol lenses for constant wear, and advised no reading.

I saw her again July 27. Her vision at that time was—right eye, 20/20; left eye, 20/30. No change in the ophthalmoscopic appearances. As she expressed it, she was beginning to see right through the spots. This case is probably one of moderate injury which will recover.

DR. D. T. VAIL, Cincinnati: The paper of the essayist is most valuable and complete. The crystalline lens has about the same strength as the ordinary sun-glass. We know what effect is produced when we focus the image of the sun on the skin by means of a sun-glass. Arguing from this, I have no doubt whatever that the retina in the macular region is burned as the result of viewing an eclipse and a miniature record of the sun permanently made there.

I had a patient, aged 76, who had cataract in both eyes, however, more advanced in the right. He gave the history that at the age of 16 he had observed an eclipse of the sun and as a consequence had noticed ever since that there was a small brown spot in the center of his field in the right eye. This spot during his seeing years impaired his vision somewhat, he stated. The left eye had no such scotoma, but was still possessed of some vision which permitted him to go about the house and familiar places. The question was: Should I operate on his eye possess-

ing the ripe cataract with the history of blind spot due to viewing eclipse of the sun sixty years before, or on the other eye which still possessed valuable practical vision? He was aged and infirm and most likely could only submit to one operation. I felt inclined to discount his remark, that there was a permanent spot of blindness from viewing an eclipse so long ago and so operated on this eye. The result was ideal from a surgical standpoint, but the central scotoma was revealed and manifested itself to his dying day. The vision at best was 20/40 and Ja. No. 3. The media were perfectly clear and he would have had 20/20 vision if the old blind spot had not persisted. Here, then, was a plain case proving the permanency of this kind of lesion, for he still presented small central scotoma *sixty years after the incident of its inception*.

DR. W. W. PEARSON, Des Moines: This short case history may be of interest because of an added feature. A young man whom I had refracted within a year appeared at my office saying an eye had suffered as a result of exposure to the sun while observing the eclipse. My record showed that he had standard vision. His complaint was that central vision was impaired. The test showed 20/20 in either eye, with the exception that he contended with one eye he must look in advance to see the letter, a symptom that we usually note in those suffering with central scotoma. His acuity of vision, etc., led me to believe that he was malingering. He had an accident insurance policy and my impression was that he was trying to collect on it. All the tests which I made, including the Peter's test, did not corroborate his contention.

DR. H. G. MERRILL, Provo, Utah: The patient whom I saw had considerable disturbance of vision, though in one eye only, as he closed the other while observing the eclipse. His central scotoma gradually diminished in size, but some of it still persists, and possibly may never entirely clear up.

DR. H. V. WÜRDEMAN, Seattle, Wash.: The warnings in the newspapers prevented many cases. In twenty miles around Seattle, on account of cloudiness, we hardly knew there was an eclipse; to my knowledge not a case occurred in the states of Washington or Oregon.

DR. JOSEPH S. LICHTENBERG, Kansas City, Mo.: These cases also occur among electrical workers and in factories where the workers are looking into a bright light, and among actors looking into "spotlights." It causes the same symptoms as the eclipse blinding.

DR. JACKSON (closing): These responses are from about 200 physicians and oculists. The cards were sent to about 600, which is not one-tenth of those engaged in eye practice in the region of the eclipse. Thus, with seventy cases, we might assume that 700 cases occurred in connection with this eclipse. Some of the reports referred to the fact that the newspapers had called general attention to it. In Denver such was the case; and Dr. Howe, the astronomer, who had been lecturing on the eclipse, spoke of it. But what saved the most eyes was the cloudy day. In Denver, and in most of Texas and Oklahoma, the eclipse was invisible on account of the clouds. But with 700 cases of impairment of vision, of which a considerable proportion will be permanent, it is of sufficient practical importance to engage our attention, and in the future this subject should be considered with regard to prophylaxis.

ULCER OF THE CONJUNCTIVA

GEORGE L. STRADER, M.D., CHEYENNE, WYO.

I wish to present this case, which is interesting, because of the difficulty in making a diagnosis. The patient is my associate, Dr. Decker. Three weeks ago he awoke one morning with his eye smarting and inflamed. The conjunctiva of the lower lid was congested and inflamed with a beginning chemosis of the ocular conjunctiva. The next day a superficial ulcer had developed about the middle of the conjunctival lower surface of the lid. The preauricular gland and one below the ear were tender and slightly swollen. The ulcer was touched with 8 per cent. trichloroacetic acid each day, with some improvement, though the chemosis and glandular swelling increased. After five days he consulted Dr. Gifford of Omaha, who cauterized the ulcer with nitric acid. An examination for spirochetes was negative, as also was a Wassermann test. He returned home and I continued treatment, using pure trichloroacetic acid for five days, at which time the ulcer was nearly healed, though there was marked induration of the lower lid and the chemotic conjunctiva covered half of the cornea. He had an afternoon temperature of from 99.5 to 101 F.

No treatment of the ulcer was given during the time intervening from Thursday to Monday, when he went to Denver where a dark field examination for spirochetes and also a Wassermann test were made, both of which were negative. A bacillus similar to a Vincent's was the only kind of bacteria discovered. When he returned home the ulcer had extended to such a degree that nearly the entire surface of the lower lid was involved. Treatment with pure trichloroacetic acid was again resumed; rapid improvement during the last two days. (The patient was examined by the physicians present.)

I wish to add that the photophobia, which at all times has been marked, is 50 per cent. better than it was two days ago. Two days ago the lid was so swollen the skin was shiny. Thinking possibly there might be pus, I made several incisions without finding any. Since then there has been steady improvement. It might be well to add that trichloroacetic acid is specific for Vincent's bacillus.

DISCUSSION

DR. H. GIFFORD, Omaha: When I saw this lesion it was about one-fourth as big as it is now, and I was impressed with the amount of infiltration in comparison to the size of the ulcer, and there was a comparatively small swelling of the gland. I had never seen a chancre on the conjunctiva, but this seemed to answer to the description.

DR. W. W. PEARSON, Des Moines: I regret to say that I cannot answer the question as to what the disease is, but merely wish to sound a word of warning on a recent experience which I had. It was a tarsitis. There was a little local inflammation. I had a Wassermann test made which was positive. I returned the patient to his physician and saw him six months later when he had an enormous malignant orbital involvement. The orbit was eviscerated and he has a recurrence at this time. I think we should be careful in the case presented today not to deny that it is a specific involvement. Another case I saw recently seemed to be merely an acute infection of a meibomian gland, but closer examination showed it to be a specific tarsitis. The appearance of a process of this type in a "specific" does not necessarily mean that the process is due to syphilis.

DR. JOSEPH S. LICHTENBERG, Kansas City, Mo.: It makes me think of the possibility of the Mikulicz syndrome.

DR. M. FEINGOLD, New Orleans: I suggest an excision for histologic examination.

DR. MELVILLE BLACK, Denver: I had the pleasure of seeing the doctor several days ago. While no spirochetes were found, this does not mean that there were none there. The debris might not show the spirochete. I am not positive that the serum from the part was examined. The appearance here is that of a primary sore. I think I should give a good stiff dose of arsphenamin (salvarsan) and depend on the clinical results. If it clears up under this treatment it will be as positive a diagnosis as we could get. It is too early for a positive Wassermann reaction.

DR. STRADER: Do you think a primary sore would flare up in twelve or fourteen hours until we had a chemosis that is holding down the eyelid?

DR. BLACK: You might get in the eyelid as much chemosis as in the prepuce, for instance. You know a very slight thing, like a little infection, say a sty, will set up a great lid chemosis. If I were you I should give him a good big dose of arsphenamin (salvarsan) and await clinical results. It is one of these borderline cases in which it is difficult to say what it is. But on account of the history and the clinical appearance I should be inclined to place this as a primary sore and deal with it as such. He might wait for secondary manifestations. Would you be willing to wait? I should not. If we can get in the arsphenamin (salvarsan) before the secondaries appear the patient has a much easier time.

DR. H. G. MERRILL, Provo, Utah: It might be suggested that neoarsphenamin (neosalvarsan) intravenously acts as a specific in Vincent's. For this reason, immediate relief after neoarsphenamin (neosalvarsan) would still not prove the case to be of syphilitic origin. Its use in this case, however, should be recommended.

CONJUNCTIVAL GROWTH

V. B. FISCHER, M.D., BOULDER, COLO.

The case presented was one in which the history of the patient was brief. The first evidence of the growth was seventeen years ago when the mother noticed a small dark-colored mass which first appeared on "the white part of the eye." Two years ago the growth was first seen by the author. At that time the growth was 3 mm. in diameter and about 3 mm. from the limbus. The boy was advised then to have the growth removed, but as it was not bothering him, nothing was done. Two weeks ago the boy appeared before the draft board. The growth was engaging on the cornea. In two years it had progressed 3 mm. to the cornea and 1 mm. additional over onto the corneal substance. At the time of the draft board examination the vision in this eye was 19/20; at the present time it is 19/30.

DISCUSSION

DR. FEINGOLD: How is the ciliary body and the lens?

DR. FISCHER: The lens is clear (mydriatic was used). There is no evidence of a tumor mass springing from the ciliary body into the vitreous or posterior chamber, and no vitreous opacities.

I am particularly anxious to know what we should do here. Shall we wait and observe the eye after we have resorted to a superficial shaving operation or shall we enucleate?

DR. M. FEINGOLD, New Orleans: The history of the case is one of a congenital pigment nevus of the conjunctiva bulbi. As is often seen in these cases the condition remained stationary for a while and subsequently began to grow. The question to be settled in the case is: Is the condition still benign, or has it already, as is so often seen, assumed a malignant character? The pigmented area in the conjunctiva is fairly movable over the underlying sclera, a condition to be interpreted in favor of the nonmalignant nature. In the center of the pigmented area, a dark brown and deep seated spot is to be seen which may be the seat of the original pigmentation in the episclera, possibly at a point of an anterior ciliary vessel. The one fact that makes one suspect the recently developed malignant character of the condition is that the patient's vision has recently deteriorated. Since mediums and fundus are to all appearances normal, such deterioration of vision may possibly be explained by the changes in the suspensory ligament through an invasion of the ciliary body by the assumed malignant process or, in a later stage, through absorption of the lens from an enlarged and invaded body. In this respect it would be useful to examine the refraction of both eyes with and without a cycloplegic. If no marked anisometropia exists, the deteriorated vision of the one eye will decidedly speak for malignancy. It might also be useful to test the vision with a pin-hole disk. Improvement of vision under these conditions will decidedly speak for malignancy, because it

would prove monocular alteration in the lens in a young individual. About seven years ago a woman called at our clinic complaining that vision of the right eye had been less good for the last two weeks. Vision was 5/12; pupil was slightly wider than that of the left eye, but perfectly reacting. As the cause of this a sarcoma of the ciliary body was found in the lower temporal quadrant after dilatation of the pupil. Enucleation was done a few days later and the histologic examination showed that the lower temporal portion of the lens was entirely absorbed by the pressure of the growth. It might also be advantageous to carefully examine the periphery of the lens under homatropin and to see what transillumination may show up.

DR. JOHN GREEN, JR., St. Louis: It seems to me that radium might be tried for a time. If improvement were not prompt, I would operate. I would excise completely, then cauterize, and allow the area to granulate.

DR. T. W. WEAVER, Wichita, Kan. (presents case): I wish to show a specimen that Dr. Feingold has just described. This is not his case, however.

DR. F. R. SPENCER, Boulder, Colo.: In these cases we have one thing to deal with, the metastasis. In Colorado two or three patients have died while the physician was waiting to decide to take out the eye.

DR. FEINGOLD: As soon as malignancy seems to be reasonably sure enucleation must be done; nothing less will do.

A CASE OF TUMOR OF THE OPTIC CHIASM

FREDERICK STAUFFER, M.D.

SALT LAKE CITY

Family History.—Chas. L., aged 10 years, weighed 96 pounds. His father, aged 50, was a physician; a periodical drunkard. He frequently left home and associated with other women. His mother, aged 37, is irresponsible; she has a wonderful imagination which she is always ready to express without much regard for truth.

Personal History.—Charles was the oldest of four children. He has always been physically overdeveloped for his age, and mentally very bright in some directions. Phenomenally bright in mathematics; being able to do complicated problems in mental arithmetic. He was always unreliable and untruthful, and developed criminal habits early. In December, 1916, his father deserted the mother and the four children. Charles was sent to an orphanage, but because of immoral and criminal tendencies he was turned over to the Juvenile Court. His sexual organs were fully developed. He was always trying to fondle women when in their presence. When he was 8 years of age he was accused of setting fire to their home and causing the death of a younger brother. He had periodic blind spells all his life; but during the last three years he was almost totally blind in both eyes, having only light perception in the right eye and perception of hand movements in the left at 6 inches.

Examination.—The ophthalmoscope revealed a perfectly white disk in both eyes; retinal vessels were normal, no signs of previous choked disk. Syphilitic history was negative. The Wassermann test was not taken. A roentgenogram gave no satisfactory clue.

Diagnosis.—The attending physician made a diagnosis of tumor of the pituitary gland.

Operation.—On Sept. 22, 1917, under ether anesthesia, I opened the floor of the sella turcica through the nose by the Hirsch method. On penetrating the anterior, lower wall of the sella, a dense fibrous mass presented itself. An attempt was made to remove some of it with a curet and alligator forcep, but without result. While manipulating the growth the boy suddenly stopped breathing. He was resuscitated and a second attempt was made to remove some of the growth, when the respiration again failed him and despite all our efforts the boy died on the operating table.

The mother was present and seemed quite pleased with the result; and when at the necropsy I found the condition as represented in this specimen, I also congratulated myself on the result.

Necropsy.—The necropsy showed that the sella turcica had been punctured in the medial line in the floor, but instead of finding the pituitary gland, its place was taken up by a tumor of the optic commissure, which through pressure downward and backward had destroyed the gland as well as the posterior wall of the sella; there remained but a slightly concave smooth surface instead of the usual saddle.

As you will see by the specimen, there is a hard fibrous mass uniformly infiltrating the commissure, optic nerves and tracts. This is probably a sarcoma. I am presenting the case because it may give some clue to further study of the functions of the pituitary gland. In this case there has probably been an entire absence of the gland for several years.

The question of greatest importance in this case is whether the condition of this boy was due entirely to the absence of the pituitary gland, or whether heredity played an important part in his case. You will observe that the pineal gland is normal.

TRANSACTIONS

OF THE

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THE DESTRUCTION OF THE PHYSIOLOGIC FUNCTION AFTER OPERATIONS ON THE NOSE AND THROAT

WOLFF FREUDENTHAL, M.D.
NEW YORK

It is not the aim of this paper to give a description of the untoward results of operations similar to the one given several years ago by John Mackenzie on the massacre of the tonsil. Its purpose is rather to induce the rhinolaryngologist to consider the physiology of the upper air tract more than has been done during the last twenty years of successful nasal and pharyngeal work and to keep in mind the bad results following procedures that are considered nowadays perfectly legitimate and necessary.

PHYSIOLOGY OF THE NOSE

A great deal has been written on this subject, but we have not arrived at the point where we can say that a universal agreement has been reached on some of the most important questions to be considered here. The writer is pleased to note that some of the fundamental principles laid down by him are being gradually recognized. In order to understand the complaints of our patients, or in other words, the pathology, we have to turn to the physiologic functions of the nose and investigate the requirements of normal respiration through that organ. The old story that the nose has to filter, warm and moisten the air holds good today. To accomplish that, Nature has provided many devices. The interior of the nose does not represent a straight tunnel, but a complicated structure preventing the air from passing in a straight line to the pharynx. A variety of bones and cartilages covered with an important mucous membrane has been set up in order that the air should strike this, thus enabling it to become physically adapted for its work in the lower portions of the air passages. Just as the food is prepared or rendered digestible by the act of chewing, salivation, etc., in the upper digestive tract, so the air is prepared in the nose, in order to be assimilated, as I have called the process.

The process of *filtering* inspired air is probably the most important function. In this respect the epithelial cells of the mucous membrane are probably a primary and essential factor

of our resistance against disease. Owen Paget¹ even believes that the epithelial cells of the turbinates and nasal sinuses have a special capacity to form "antibodies" against tuberculosis. An intact mucosa will therefore mean a great deal for the filtering function of the nose.

The second requirement of *warming the air* is so easily understood that it need not be discussed here at length. As Aschenbrandt and R. Kayser have proven long ago, the air is heated to 30° C. in the nose, no matter what the outer temperature be. So here again it must be repeated that the mucosa, and if possible an intact one, is essential for the work to be done.

Much more difficult is the third task, namely, of *moistening the inspired air*. This question, to which the writer has given much thought and study for many years, is, it seems, not universally appreciated by physicians. The idea still exists in some quarters that this is rather an unimportant factor, that it can be accomplished by the mucous membrane under almost any circumstances and in nearly every kind of environment.

AUTHOR'S OBSERVATIONS

As early as 1895 I tried to establish the facts that (a) a dry mucosa and more so an atrophic one, is never able to moisten the inhaled air satisfactorily, and (b) that the air, to be breathed in, must contain a certain percentage of humidity in order to be easily "assimilated."

In proof of these theories many points were brought out in that year and during the following ones. Suffice it to say here, that as disclosed by many investigations the fact was established that the dwellings inhabited by us and in which we often spend 23 hours of the day, do not contain nearly enough humidity for physiologic respiration. When the air in our rooms is nearly dry, as it frequently is, it desiccates the mucosa, thus causing pathologic changes to be discussed later on.

The normal mucosa of the nose is capable of overcoming the lack of moisture in the air to a certain degree only. Beyond that it dries out itself and can no longer be a factor in assimilating the inhaled air. Thus a rhinitis sicca is produced with or without a corresponding condition in the pharynx and nasopharynx. A number of these cases finally result in atrophy.*

* As to the development of atrophic rhinitis in this climate (namely, Denver), the opinions of two prominent rhinologists seem to differ. While Dr. Gallaher believes that this is rarely the case, Dr. Robert Levy expresses a contrary view. (Transactions Am. Otol., Rhinol. and Laryngol. Soc., 1915, pp. 188 and 189.) But do these two gentlemen mean the same condition in speaking of atrophic rhinitis as I do? And do they agree among themselves about that disease?

This process of desiccation that has been observed in other parts of the organism as well, has been named xerasia by the writer, while Sticker, who independently came to similar conclusions, called it xerosis.

OBSERVATIONS BY OTHER WRITERS

In distinct contradiction to these findings of mine were certain studies of the effect of various atmospheric conditions on the upper respiratory tract, a work to which the prize was awarded by a national society.² The author's conclusions, as just mentioned, are entirely different from my own. By analyzing his paper, however, so much will be found that is based on erroneous experiments and deductions that his work cannot be considered conclusive or final.

First, the exposure of any person for a short period to the atmosphere of a room and then changing to another with different atmospheric conditions will give rise to only acute symptoms, but never will it produce a rhinitis sicca which is brought about by many months or probably years of living in vitiated atmosphere, that is, vitiated as to the lack of humidity.

Some of those examined by the author of these experiments showed on examination an enlarged turbinal, others redness or the reverse, again others increased moisture, etc. But what does all this prove? Absolutely nothing. These symptoms may or may not change, when the condition becomes chronic. Besides the individual susceptibility to drafts, exposures, etc., had not been tested beforehand, so that a good deal of the value of these experiments is lost for this reason alone. Furthermore, there is a great difference of findings in different seasons of the year, as pointed out by M. Behr³ and the writer. During the month of February, for example, you will find many more cases of rhinitis sicca than in August. Such great variations ought to have some effect on experiments. Then the assertion that some cases showed increased or decreased moisture brings up the question: How was the moisture measured? By the Glatzel method? Certainly few physicists would consider that conclusive.

But the strongest point against my deductions was made by this author in the statement that laundry workers, who spend most of their time in a moist atmosphere, "showed by far the largest percentage of cases of atrophic rhinitis of any of the group." This puzzled me only until I found an explanation for the mistake made in the above article. On page 166 (l.c.) it says:

"Investigation of the laundry plants shows that the steam comes directly up into the faces of the workers standing near the mangles, washing machines and other appliances much of the time. In fact, we feel certain that these steam laundry workers are far more exposed to high degrees of humidity than the figures indicate."

It appears as if the last sentence was written in support of the author's theory or supposed theory that a great amount of moisture in the air is rather conducive to atrophic rhinitis, for among thirty-three laundry workers examined he found twenty-one suffering from that disease, or 63 per cent. But if one examines these statements more closely, what do you find? You find that "the steam comes directly up into the faces of the workers . . ." and naturally up into the nose. And what happens when steam at a high temperature is brought in close contact with such a delicate organ as the nasal mucosa will be easily understood. It simply destroys the membrane or burns it. The skin of the face may or may not withstand the insults of the hot steam—the author does not mention anything about that point—but the nasal mucosa gradually undergoes atrophy. Place those laundry workers wherever you please in absolutely dry or absolutely moist air, the result will always be the same—atrophy.

In the second table under this heading the author shows the average atmospheric conditions in a large number of boiler and engine rooms. Surprisingly to say, according to his findings, the average relative humidity was only 26.5 per cent. In other words, it was only one-half of what he himself had designated as normal (50 per cent.). Therefore, we have here a deficiency of humidity in the air and not a surplus of it. How the author could give this as a proof of his theory is difficult to understand. On the other hand, this is exactly what I tried to prove long ago, namely, that the lack of sufficient moisture in the air is exactly what produces a rhinitis sicca and finally a rhinitis atrophicans.

COMMENT

It is not a pleasant task to scrupulously analyze somebody else's work. I hope, however, it will be understood that this is not at all a personal matter, but one of greatest importance not only in regard to heating and ventilation, but more so as to the prevention of a number of nasal affections with all their sequelae. It is my claim that during the winter months people suffer from lack of indoor humidity, and it is not a matter of indifference if that is denied by others.

This question has commenced to attract the attention of the medical and lay public. But even in the minds of such a distinguished body of men as the editorial writers of the *Journal of the American Medical Association* are, there seems to be confusion. One editorial states (Oct. 6, 1917, p. 1174): "The question as to whether the atmosphere can ever become too dry for comfort or physiologic wellbeing has not yet been satisfactorily answered." It seems to me that I answered that question long ago in the same journal.⁴ But in spite of the above sentence the editorial continues: "In cold weather the moisture may be largely precipitated from the air so that it becomes extremely dry when it enters houses. This led to the desire and the practice to moisten such air." What a contrast between these two sentences.

Along the same lines runs an article by Frederic S. Lee,⁵ in which the following is found: "The harmfulness of living in confined air is found in certain physical rather than chemical features—the air is too warm, too moist and too still; and if it has not these physical features it is not harmful." Too warm may be true, too still may also be correct; but too moist is a grave mistake. I would like to see any ordinary living or office room, when the outside temperature is below the freezing point, that shows by actual measurement the presence of too much moisture in the air. I wonder how many dwellings Dr. Lee has examined to that effect. In all my investigations the humidity was reduced to such a low degree that its absence showed distinct pathologic effects on the mucosa of the upper air tract.

It should be mentioned here that in 1916 Dr. S. Josephine Baker⁶ made investigations in cooperation with the New York State Commission on Ventilation as to the value of certain kinds of ventilation, etc. As these studies have something to do with our theme, the conclusions arrived at by Dr. Baker may be mentioned here.

"Children in classrooms with closed windows and ventilated by mechanical methods were more subject to respiratory diseases, severe enough to keep them from school attendance, than were children who were in classrooms kept at the same or lower temperature and ventilated wholly by open windows." And then again:

"Children in classrooms with closed windows and ventilated by mechanical methods were more subject to respiratory diseases not sufficiently severe to keep them from school attendance than were children who were in classrooms kept at the same or lower temperature and ventilated wholly by open windows. The rela-

tive humidity of classrooms, whether ventilated by natural or mechanical means, was not a causative factor in the occurrence of respiratory illness among school children."

It is especially the last paragraph that deserves our attention. Such a broad statement as this has to be proven, but we looked in vain for any proof. Later on Baker says: "It seems impossible to show any relation between the percentage of absence and the relative humidity and saturation deficit." Of course not, such a thing could not be proven at once. One cannot say, that on account of a saturation deficit fifty or 100 pupils are absent today or will be absent tomorrow. Such influences work slowly, as mentioned in the foregoing, and they may not be noticed until many months have elapsed. Further on Baker remarks: "Where the relative humidity was low, one class with 21 had an absence percentage of 6.8, while another with a relative humidity of 28 had an absence percentage of 31." We hope the author does not consider 28 a high relative humidity; the lowest normal is 40 per cent., and it is only above 40 that human beings feel comfortable (of course, up to a certain point only). But from these quotations it can be seen that no conclusions should be drawn from such findings. Experiments have to be carried on differently when the question of moisture is involved.

MECHANICAL MEASURES INVENTED

To overcome this deficiency of moisture many a device has been recommended. P. W. Geldsbury realized the importance of the problem of constructing buildings in such a way as to keep the interior up to a fair degree of humidity. But he thinks that so far engineers have made little practical progress toward its solution. Their ingenuity has been taxed to improve the moisture in large public buildings. The devices for this have so far proved too expensive for private dwellings, hotels, offices or school houses.⁷

Bryce of Ottawa, who very likely had a similar experience, says that 75 gallons of water must be evaporated daily in an ordinary-sized house to maintain reasonable humidity "under the conditions of our northern winter." It seems plausible that this statement holds good for New York as well. But of the many devices proposed for such a purpose we cannot recommend any. Even the four radiator devices as tested by E. P. Lyon at the University of Michigan are "practically worthless."

CLIMATIC CONDITIONS SIMULATING OPERATIVE
INTERFERENCE

The question of humidity has been discussed here at considerable length and you may justly ask, for what reason? The reason is that our indoor life, especially in winter, and the lack of sufficient indoor humidity, brings about certain symptoms that frequently seem to be a strict indication for operative intervention, but are not in reality.

CASES CITED

Let us illustrate this by a few examples.

A. P. comes into my clinic complaining of his nose being stopped up. On examination the right side was found to be wide, almost atrophic and very dry. The left side was clogged by the septum being deviated toward the left to such a degree that there was only a narrow opening. The mucous membrane here was in quite a good condition. When the patient was asked, through which side he could breathe the better, he promptly answered: The left one, that is, the narrow side. I recollect the remark that someone made: How foolish the answers are even from apparently intelligent people. Well, the patient was operated on and his septum straightened out with an excellent immediate result. Soon afterward he returned, however. He complained bitterly saying that while formerly he could breathe at least through the left side, now that side was gone too. Crusts were found there and the mucosa did not show the same healthy appearance as before the operation.

Another example: J. D. feels a dryness in the throat, especially so in winter. He has been operated on many times and (as a result?) has lost his sense of smell. "Since my first operation I feel no air. Plenty of room." This man's nose is characteristic by the absence of both inferior and middle turbinated bodies. The septum had been straightened, and "what else is there to operate upon?" he asked me. The nasal air passages were large and the mucosa dry.

In the first instance there was a tendency toward atrophy that was evident on the right side. No sooner had we operated on the other side, than the same process set in there, destroying the physiologic function of the mucosa on that side as well.

In the second case the nasal passages were made so wide that the air reached the pharynx in a direct line. There was no possibility of assimilating the air to the needs of the organism and

the patient felt a real hunger for air. (A similar experience was reported by Dr. Weil² of New Orleans.)

A colleague in this city had suffered from nose trouble since childhood. Tonsils and adenoids were removed in childhood. He became a physician, but still suffered, and some turbinates were taken out by a specialist. Not feeling relieved he consulted another laryngologist and this one said: "Your turbinats were removed? Why, they are immense." Again they were operated on, and a year later a third time. Thereafter he became hoarse easily and was again examined by three different laryngologists, consecutively, each one of whom declared, independently of the other, that he had a sigmoid shaped septum and that his voice could never improve without the septum being straightened. That was done and now he is worse off than ever before. His mucosa "swells" at the slightest provocation, so that he is unable to breathe. He suffers from hoarseness, and in short feels miserable. While writing this another colleague consulted me with a very similar story.

These cases and many others observed later on, made me pause to consider in how far we are justified in performing certain operations. It was clear to my mind that we had gone too far in both of these operations, and that the patients' complaints were due to climatic conditions and not to an easily perceptible faulty formation of the septum or the turbinats. If simpler methods had been used the patients would have experienced relief very soon, which is impossible if a real bony or cartilaginous obstruction is the cause of the trouble. Besides the tendency toward atrophy should always be kept in mind, especially when the initial stage is present already.

FURTHER CONSIDERATIONS

This brings us to the question: Is an absolutely straight septum normal? There was an era in rhinology when it was thought that at least the aborigines had straight septa. In 1904 I examined⁸ more than 800 crania of aborigines and was surprised to find that more than one-third showed abnormal conditions of the septum. Other observers reached the same result and it was pointed out that asymmetry of the organs is probably the most normal condition. Consequently many rhinologists have learned the lesson that *there are nonobstructive deviations which do not necessitate an operation.*

REFERENCES IN THE LITERATURE

In this connection an article by Dr. H. M. Goddard⁹ of Philadelphia is very interesting and instructive. While he admits the many beneficial results obtained by a submucous resection he lays great stress on the importance of an intact nasal mucosa. "By destroying the mucosa we are very apt to substitute a perverted respiratory function for an obstructed one." Goddard then describes a series of conditions that often do set in afterward, produced by the lack of epithelium, etc., symptoms which I ascribed to the impossibility of assimilation of the inspired air. This is not the place to give the absolute indications for submucous resection, an operation that the writer has to perform quite frequently. But it may be the time to warn colleagues, that not every deviated septum spells operation.

THE AGE OF THE PATIENT

This is another point of importance that has to be taken into consideration. In older or middle aged persons, that is beyond the age of 45 or 50, the organism has adjusted itself to a certain amount of pathologic changes to such a degree that it does not require any operative interference. Such people with a deviated septum or a large spur often feel perfectly comfortable, as they do not need as much air for breathing purposes as in younger and more active days. They have unconsciously regulated their habits and their daily work to fit into this altered condition, and a change is not required for their well being. Let us illustrate this by an example taken from another field of our work. As is well known the glottis in bilateral paralysis of the abductor muscles is narrowed down to a very narrow slit. A minute amount of air can pass through this, especially since at each inhalation the glottis paradoxically closes up more. Yet such patients may live for years in relative comfort, if they learn how to get along with little respiratory pabulum. I distinctly recollect one patient, who was under my observation at the Montefiore Hospital for more than eight years. He had that condition of the abductor muscles due to tabes. Besides, his lower extremities were paralyzed and he was totally blind. He had to be rolled around in his chair and had very little occasion to exert himself. He did not need much air for his daily requirements. That was in the same proportion as the intake of his food, which was much less than in the days of his activity. (Parenthetically, it may be mentioned that another patient with the same condition in his

larynx, but without any affection of his lower extremities and eyes, was walking around, became drunk one day and was found dead on the street.) In making routine examinations¹⁰ on several hundred patients a number of cases were found that were similar to the one just narrated. Others have observed the same conditions in cases in which they obtained no history, as for example those reported by Dr. W. B. Chamberlin of Cleveland.

Similar obstructions, though not so exaggerated, may prevail in the nose for many years with impunity. I have seen such people reach an old age experiencing not more discomfort than others who had been operated on. And these discomforts were easier to bear than an atrophic rhinitis or a rhinitis sicca.

THE TURBINALS IN SINGERS

What has been said in general about the septum narium holds good for the turbinals to a still greater degree; that is, among ordinary mortals, but how about singers? Since nothing was found in literature touching that subject, it may be timely to give my experience with two well known singers.

CASE 1.—Mr. X. complained of some difficulty in breathing. Both inferior turbinals were removed by a rhinologist of standing, whereupon Mr. X. was unable to sing for one year and a half.

CASE 2.—Mr. N. N. sang one night in one of the European capitols with marvelous success. The next day the turbinals were removed and from that moment on he was lost vocally. He could not produce any high notes and never again appeared on the stage.

These cases are reported as they are known to me personally. Are they exceptions or have others observed similar effects?

On the other hand, some singers may and do gradually learn how to place their voices and accommodate them to the altered anatomic relations. But before operating one should never forget the double connection the nose has with the voice—as an air passage and as a space for resonance. The air on passing by the numerous projections based on the principle of an increased surface is adapted for the requirements of the system. The integrity of these hygienic functions of the nose is of special importance for the voice producing apparatus—the larynx.

The importance of the nose as regards resonance depends on the pneumatic cavities and on the conchae with their intact mucosae. It is wise to keep that in mind before removing any of these important tissues.

THE ACCESSORY SINUSES

As far as the ethmoids are concerned the idea of Dr. Holmes has to be mentioned that in all probability the cellular structure of the ethmoid labyrinth had for one of its functions the protection from cold. That has been the writer's experience in a few cases of ethmoid operation, but more so after radical operation on the antrum.

As to these radical procedures on the accessory sinuses, Dr. E. B. Gleason¹¹ says:

"I cannot help feeling that the status of radical operations on the nasal accessory sinuses is worse than that of the radical mastoid operation, which is, of course, sometimes necessary to save life, but which, in its final results, sometimes leaves a less satisfactory condition than if no operation had been done, and sometimes requires much after-treatment, from time to time, to prevent recurrent suppuration. In other words, one pathologic condition has been substituted for another. . . ."

AUTHOR'S SUGGESTIONS

In bringing these data from other observers as well as my own experience it is not the aim to discourage entirely operations on the nasal sinuses, some of which are directly life saving, while many others have offered great relief to suffering patients. It is done rather with the idea not to place all your confidence in these operations, thereby neglecting other means of relief. A normal mucosa is apt to overcome many difficulties, probably even an infection of the sinuses. To keep it normal or bring it back to the normal, the deficiency of indoor moisture will have to be combated first. Parenthetically, it may be added that one of the effective therapeutic measures in chronic empyema of these sinuses is the use of negative pressure (vacuum pump), as advocated by Haskin, Coffin, Harmon Smith, W. A. Wells, MacWhinnie and lately by Gleason. It acts merely by producing Bier's hyperemia, and hyperemia of the mucosa means nothing more than increased influx of serous exudation, or, in other words, increased humidity.

THE OPERATIONS ON THE THROAT

While in certain affections of the nose the pathologic conditions may be overcome by putting the patient under normal hygienic conditions, as outlined in the foregoing, it is somewhat different in the mouth and throat. I have proven elsewhere¹² that the lips, the teeth and the tongue are affected by the lack of indoor

humidity, but so far I am not able to prove to my own satisfaction that diseased tonsils and adenoids are the result of such conditions too. Yet we have to operate on them and do so frequently.

ADENOID VEGETATIONS AND TONSILS

From their removal no untoward results are known to me, unless they were due to faulty technic. But how about the tonsils? Is it true that indications for the removal of tonsils are not as simple as they appeared to be? Does the general practitioner or even the laryngologist always know when to operate and when not? Somebody has remarked, that it was easier to remove a tonsil than to know whether or not it should be done.

What has happened since Dr. John A. Mackenzie¹³ wrote his article on *The Massacre of the Tonsil*, in 1912, in which he speaks of "reckless, ruthless and unnecessary" enucleation of the tonsils in children?

This article, on account of its forcible language and of the prominence of its author, made an impression all over the country. Formerly schoolchildren in the poorer districts of New York City were driven by the hundreds to the clinics, whether the parents gave permission or not. And many of these children were operated on with or without good reason. The nurses employed by the New York Board of Health seemed to glorify in taking as many children to the clinics as possible. On some afternoons I was confronted with from twenty to thirty of such children of that category, and on many of them I refused to operate to the apparent disgust of the nurse. That practice has markedly diminished.

Some people still persist in telling you that since tonsils have no functions to perform, it is best to remove them and to do it radically. I hope not many thinking physicians will be found to subscribe to such theories. For in the first instance, it is perfectly absurd to advise removing an organ from the human body because its physiologic function is as yet unknown. Secondly, however, the tonsils have functions to perform. They have to lubricate the food that is taken in, thus performing the first and perhaps very important step in our digestion. Secondly, they have to lubricate the throat during the act of speaking and singing and are of special importance to speakers and singers. Furthermore, the mechanical significance of the tonsil has to be considered (Kenyon). That part will be taken up shortly. Finally, the tonsils have the same task as the nasal mucosa, only to a greater degree. They, like all the other lymphatic structures

in the pharynx and elsewhere, have to ward off dangers by inhalation, infection and especially here by contaminated fluids of the mouth and pharynx. They do most important filtering work for the organism and with their phagocytes take up the primary battle against the tubercle bacillus. "Remember," says Dr. Henry L. Swain, in an exceedingly interesting article that just came to my notice, "it is only when the phagocytes in the tonsils fail in their work that the germ gets into the second line trench, the lymph nodes of the neck. Many times, therefore, they kill the tuberculous germs off, and it is lucky for us that they do."¹⁴ For these reasons there is a strong opinion against the indiscriminate removal of tonsils (compare Dr. C. W. Richardson's¹⁵ article and Dr. Otto T. Freer, who speaks against the "focal infectionists"). On the other hand, some men are emphatic, and the writer is one of them, against radicalism in every case, where an operation is strictly indicated. When I read a part of this paper before the New York Medical Union on May 28 a practitioner of wide experience, Dr. Daniel Cook, remarked in the discussion following, that a good deal of codliver oil and more of fresh air (taking the pupil out of school) had saved many a little patient of his from an operation. Dr. Francis Huber said he had used the guillotine for more than twenty-five years, and has had no reason to regret it. I believe that both these gentlemen are correct, but have to add that Dr. Huber speaks from his standpoint as a pediatricist.

According to my opinion, there are indications for partial removal of the tonsil and radical tonsillectomy, both of which I have practiced for years. And it is my experience that whatever is left of a tonsil if it is normal tissue serves as a protection to the organism. To prove this I may cite the following fact: After operations in the nose occasionally an infection of the throat follows, that may spread to the cervical glands. Now it has been my experience that this cervical adenitis is more frequent after a perfect enucleation, than in those cases in which part of the tonsil had been left (or naturally the whole tonsil). This is also the experience of Dr. Edgar M. Holmes, as expressed in a conversation with the writer. I do not speak against tonsillotomy or tonsillectomy. Both have their places in medical practice. But I am strongly against overoperations on the tonsils, which are unnecessary and not without danger. Physicians and laymen have begun to recognize that neither tonsils, nor for that matter teeth, are at the bottom of all evils in children, and that some tonsils may be left where nature placed them.

TONSILS IN GROWN PERSONS

In older patients their removal has always been considered from a different viewpoint. It seems that after tonsillectomy the impaired throat of the adult has less capacity of readjustment than that of the child. Yet tonsils that suppurate at certain intervals will have to be removed *in toto* so long as better methods of getting rid of the focus of suppuration are not known. Likewise, the tonsils will have to be dealt with in rheumatism, when there is positive evidence that they are the etiologic factors in the individual case. There are other indications for tonsillectomy which may coincide with those in children, but may well be omitted here.

However, a tonsillitis or a recurrent tonsillitis in the majority of instances is not a local affection, but a manifestation of a systemic condition. To remove the tonsils in such cases is like removing one of the defenses nature has set up for protection. It will be best to illustrate this by one of many examples that came under my observation.

CASE 3.—Fr. C., aged 35, consulted me last winter on account of an acute tonsillitis. His fever rose to 102.5 F., but after about ten days he was well again. A few months later he was operated on by somebody else, the tonsils being enucleated radically. This winter his troubles began very early and it was almost impossible to free him of his sore throat. Tonsillectomy had been done *lege artis*, but the cicatricial tissue that formed afterward was so abundant that a good deal of the mucous membrane was destroyed. He suffered now from a stubborn pharyngitis that bothered him until warmer weather set in.

The power of resistance had been weakened in this instance and with the fall of the tonsils fell one of the strongest fortifications, or as Hudson-Makuen put it: To remove the tonsil in such a case is like killing the goose that lays the golden egg.

OTHER DANGERS

However that may be, tonsillectomy leaves other dangers in its trail, as pointed out by Hudson-Makuen, French, Kenyon and Kradwell, Connor, Joyes and many others. Those dangers are noticed mostly in alterations of the speaking and singing voice.

The appearance of tonsillectomized throats has attracted my attention for years past. Over and again have patients consulted me showing such masses of cicatricial tissue, adhesions, loss of muscular tissue that one is astonished at the destruction brought about by the operations now in vogue and performed, not by

hospital interns, but by leading men in our specialty. Scars, where mucous membrane is needed, work havoc, as shown in the foregoing case, as well as by many other examples that every one can see who wants to see. As to the other defects following tonsillectomy, the articles by Kenyon and Kradwell,¹⁶ and lately by Kenyon,¹⁷ alone are so convincing that they are bound to change our mode of operation in the future. The work these authors have done "has failed to quiet any feeling of unrest with respect of the operation on either the speaking or the singing voice." The physiologic importance of the palatopharyngeus and the palatoglossus muscles, the mechanical significance of the tonsil, including its elaborate capsule, the question of the intrapharyngeal aponeurosis and the capsule, and many other factors have been explained so clearly by Kenyon that the damage done to the voice by injuring these parts can be easily understood. His article is based on the examination of 161 tonsillectomized throats, and "on a study of thirty cases of vocal or other disturbances resulting from tonsillectomy." Indeed an appalling number.

AUTHOR'S RECORDS

In analyzing my own records I am convinced that the number of such disturbances seen by me is still greater. This is also in accord with Makuen's experience that deformities of serious significance occurred in the hands of the most skillful operators. Consequently, one is not surprised at the remark of Kenyon and Kradwell: "It seems conservative to say that following tonsillectomy the palatoglossus and palatopharyngeus muscles are *never* wholly normal in their action." "Never" is a dreadful indictment against our present mode of operation.

In a similar way, Dr. W. E. Connor¹⁸ expresses his opinion that the voices of singers have been practically destroyed in some instances, while the functions of the tonsils, be they ever so small, have been denied these patients forever.

However, be that as it may, it is the belief of some men, that tonsillectomy serves to destroy not merely a possible function of the tonsil, but also to either disturb or destroy an important physicommechanic function.

OBSERVATIONS OF OTHER OPERATORS

On the other hand, there are men who have expressed different opinions. Among these is to be mentioned Dr. I. W. Voorhees,¹⁹ who went to the great trouble of sending out questionnaires to 500 laryngologists and 500 singing teachers. While

Dr. Voorhees' paper deserves appreciation, its statistics, like other statistics, have to be taken with a certain amount of reserve. Nothing, to my mind, is so misleading as statistics, no matter how truthful their authors may be. From his statistics, Dr. Voorhees draws the conclusion that in singers—that was the question he was interested in—there need be no special fear of bad results after tonsillectomy when done by skilled operators. Yet an analysis of some of his points does not quite seem to justify his optimism, as for example the answer to his Question 2: "Out of 341 cases only forty-six showed cicatricial contractions, which is truly an excellent operative record." That means about 12 per cent. bad results, which does not impress me as an "excellent record" for professional singers. Cicatricial tissue in the absence of the lubricating function of the tonsil is a defect that can be overcome by few singers only.

In reply to his Questions 5 and 6, ninety-five men reported no bad effects, while thirty-eight men had noted untoward sequelae in 172 cases. When one considers that these effects remain permanent in a goodly number of cases, one is impressed by the rather large number of men (38 out of 133, or about one-quarter) who experienced bad results. One-quarter is a surprisingly large number against tonsillectomy and not in favor of it.

COMMENT

It is after studying reports like these that one comes to recognize the physiologic necessity of the tonsil and tries his utmost to preserve at least a portion of this organ.

After a great deal of study and hard work in this field for the past decades the question of tonsillectomy and tonsillotomy will have to be taken up once more and investigated again from every point of view. Not only will this question have to be taken up, but many other operations on the upper air tract will have to be scrutinized with the idea of determining whether more conservative means will not serve the purpose and of preserving as much as possible the physiologic functions of the different structures.

In conclusion, permit me to say that it is time that we clean up our ranks as the gynecologists have done some fifteen or twenty years ago. They succeeded in preventing the removal of almost every ovary that came under observation. It is my hope that in the near future the same achievement will be accomplished in our field of work too.

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DISCUSSION

DR. JOSEPH C. BECK, Chicago: We are accustomed to having good papers from Dr. Freudenthal, and this is not an exception. It will remind those who were present at the meeting of this Academy in Denver a number of years ago of the contrast in this paper and one then presented by Dr. Dudley Reynolds on the "Inferior Turbinate Body," with the exhibition of 400 which he had removed, curing all kinds of diseases of the upper air passages as well as various other conditions of the body. Almost as many cures as we now read about are accomplished by the removal of the tonsils.

There was one part of his paper that impressed me. At that time the discussion was absolutely against the removal of the turbinates. Twenty-seven discussed the paper and about all condemned the procedure. Dr. Freudenthal's paper has recalled to me the paper of Dr. Cox who has done some excellent work along this line and received a prize for his work. You have all had the experience of atrophic rhinitis causing hunger for the air, and the procedure is to put back something, and we distend the septum by plastic transplantation to relieve it. Referring to the septum, more credit should have been given it for the value it is in the respiratory function as well as for drainage. That is rather underdone. Very frequently it is left undone when it should be done. I have had inferior turbinectomies done with marked benefit. I suppose this reference is to their wholesale removal.

As regards the sinuses, the intranasal operations for suppurative or non-suppurative cases must have distinct indication, and good results are obtained. But I have had many patients who came to me with obstruction and interference with drainage in whom the condition was relieved, and who had head pains and neuralgic pains, and then the dryness of the respiratory tract. I am sure too few punctures of the antrum are made to determine whether that cavity is infected. More is done to the ethmoid, and not enough to the antrum of Highmore. I know that some trouble comes; I have definite records of nine cases of fatalities from operations on adenoids in infancy, or in small, delicate children. These cases I have reported and have feared that a good operation might be done away with and a damper put on it because such a result does occur.

In regard to the other matter, I do not agree with the Doctor at all. That would take us back to something we will be sorry for if we adopt any moderation of technic. The duct is obstructed. The pediatricians know nothing about it. Ask the internists. We will not go back to that I hope. I think we should adopt a more conservative method. If we take it out I want to take it all out; but it is not necessary to take out every tonsil. I do not advocate that. But we do know these are foci of infection that do play an important part in systemic infection. I can prove that relief in some of these cases has come by drawing down the tonsil by the wire and removing this tissue by curettage and removing every vestige of epithelium and then release the instrument and let it go back. In the potential cases with symptoms not too alarming we should have the complete operation of tonsillectomy.

As to leaving a part of the tonsil and having less glandular infection, that has not been my experience. That it does occur is just that much more evidence there is a focus there and it should be removed thoroughly.

DR. S. H. LARGE, Cleveland: I think the Doctor's paper a very timely one, and I heartily agree with some of the things. I feel like Dr. Beck, "If we are going to remove the tonsils it should be done completely." I have seen a number of cases following the removal of adenoids in which a great deal of harm had been done.

We had a physician in Cleveland, and it seemed that nearly every patient that came under his care had his postnasal pharynx scraped, and we are now seeing the bad effects of this, as a large number of these patients have a great deal of scarred tissue.

I think the Gottstein curet is a dangerous instrument, as in most cases in which it is used the mucous membrane and even the deeper structures of the upper part of the pharynx are destroyed.

In the La Force we have an ideal instrument as it is impossible to damage the underlying structures when using it.

DR. ROY PH. SCHOLZ, St. Louis: The essayist in his paper did not in my opinion emphasize dryness of the nasal mucosa sufficiently. I find that patients who have a dry nose, frequently interpret their symptoms as those of insufficient breathing space in the nose; nasal obstruction with often a tense feeling or feeling of expansion in the region of the nose. I have observed this not only when the mucous membrane was dry in both nasal chambers, but better still is it demonstrated in cases in which one-sided atrophy exists (septum deflected far to one side causing great obstruction on that side, leaving the other side very roomy, the mucous membrane becoming dry and later atrophic). I find that frequently these patients complain of nasal obstruction on the wide or dry side, when in reality the dryness is the disturbing element. I believe that in these cases, one can easily experimentally prove my contention. If the dry surface be moistened and kept moistened by such lubricant as a dram of lanolin to the ounce of liquid albolene or the like, the patient is then free from his symptoms. To give this particular type of case more or less complete and permanent relief I do a modified submucous septum operation, which consists in replacing the septum to the median line, or even beyond, without the removal of tissue. I have also found in some operative cases, in which the operation has increased the breathing space on one or both sides, that during the stage of healing and before tissue compensation has taken place, when there is more or less dryness the patient frequently interprets the resulting symptoms as those of obstruction.

I agree entirely with the previous speakers in that not enough emphasis can be laid on the advisability of saving the mucous membrane in the

nose wherever possible, and avoid producing wounds which result in extensive scars. I believe as does Dr. Beck that the removal of the tonsils should not be expected to clear up all throat symptoms; too often tonsils are removed from throats generally inflamed (general pharyngitis), and after their removal the operator and patient are often disappointed to find the symptoms of which the patient complained still existing.

DR. JOHN H. FOSTER, Houston, Texas: The statement is frequently made that the tonsils are important in lubricating the food and throat. I have heard this statement made for years and should like for the essayist in closing to state what proof exists to substantiate it. Personally, I doubt very much that the tonsils contribute anything in the way of lubrication.

DR. FREUDENTHAL (closing discussion): I have learned as much from the discussion as when I prepared the paper. The 400 turbinates that were shown here will go down in history. I did not bring out all the indications for operations on the septum. I have to perform the sub-mucous resection frequently. But if we have one side obstructed and the other atrophic, the patients will breathe easier if the mucosa is moistened. But we cannot stay by them all the time with vaselin and sprays, etc. I overlooked the case Dr. Beck spoke of.

As regards the tonsils, I am sorry the doctor disagrees with me, but he has not convinced me. In an experience of twenty-five years we have done tonsillotomy many times when we did not know what else to do, and many of them after twenty years were found to be perfectly normal. How does the Doctor explain that? But I agree tonsils should stay in when they are perfectly normal.

For the mucosa of the turbinates I often use the electric cautery nowadays. The experience of Dr. Large in his own family is very interesting. Where you have simple hypertrophic tonsillitis so that the little patient cannot breathe, these tonsils should be clipped off. That Dr. Beck has no scar tissue is a great exception. I do not believe there are many patients operated on in New York City who do not show enormous scar tissue. Your cases from Chicago do not come to me, apparently.

As to the question of Dr. Foster, I have to refer him to the publications by Swain, Kenyon and others.

THE MODERN MASTOID OPERATION

JOHN J. KYLE, M.D.

LOS ANGELES

There is nothing especially new about what I have to present on the subject of mastoid operation. It is like taking down an old dusty book one has read many years before — the characters are as fresh to the reader's mind as though the book were just off the press.

EARLY HISTORY

It is very difficult for one who reads of the early methods of centuries ago, to find the exact name of the first man who conceived a mastoid operation. No doubt but in the time of Hippocrates, there was a superficial knowledge of disease of the ear. Ambroise Paré, the greatest and most gifted surgeon of his time, was probably the first surgeon to do the mastoid operation in France. In the history of Catherine de Medici by Balzac, the author especially speaks of Ambroise Paré as doing such an operation at the siege of Metz. Balzac had the reputation of knowing the literature of his time before beginning a book. Kuster and Schwartze are credited with doing what we now call the modern operation, that is, the modern methods of obliterating the cells by first approaching the antrum. Schwartze, in 1868, did the first mastoidectomy and antrotomy, and since then others have made additions to the original operation, that is, the closure of the wounds after the operation and postoperative treatment.

From Schwartze's operation it was a far step to the radical operation. The radical operation evolved by Schwartze-Kuster-Bergamn-Stack is now the recognized operation and came into vogue as late as 1891. It was performed with or without myringotomy and ossiculectomy. As early as 1879, Schwartze and Kuster advocated a mastoidectomy and antrotomy and removal of the posterior wall.

I remember attending an otological congress in Boston a few years ago. Mr. Heath of London was present and told of his method for the conservation of the remaining ossicles and drum membrane. The method he described was declared to be unscientific and one English physician arose and remarked that the English otologists did not countenance the Heath operation. Since that time hundreds of operations on the mastoid with a

view to the conservation of the ossicles, have been performed, and the results have been in most cases a success. Heath's operation was practically the Schwartze-Stack operation without myringotomy and ossiculectomy, and with certain methods of closure.

Kuster was the next to emphasize the fact that in the case of middle ear disease if the radical operation was performed when the bone cavity is filled with pus, the middle ear must be wholly exposed and drained perfectly. This portion of my subject is especially under consideration in this paper.

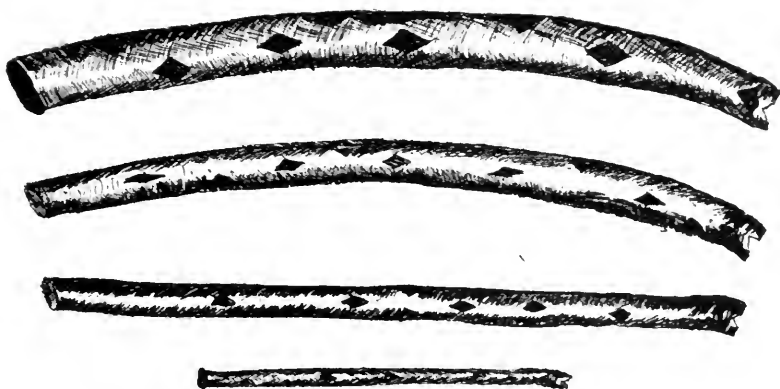


Fig. 1.—A series of rubber tubes for simple or radical operations. The idea of some of these was taken from the work of Dr. C. A. L. Reed of Cincinnati.

CAUSE OF POSTOPERATIVE FISTULA

Many cases of persistent fistula following a simple operation will close and fill all over again. However, if a fistula persists it suggests that the cells of the antrum have never been properly exposed and the cells completely obliterated. Sometimes the operation approaches the antrum too far back and below the spine of Henle. It is interesting to note in a large number of cases how the important landmark of Henle varies in individuals. It may be almost absent or stand beak-like on the posterior and superior wall. Many surgeons totally overlook the necessity of locating the landmark in approaching the antrum, and then the antrum is something never found.

There are more cases of mastoiditis in the Army than in civil life, especially among recruits. The number in a ward in some cantonments makes one think that the disease is almost of a contagious nature. This is due, however, to the contagious and infectious organisms that exist in a healthy person in the post-

nasal space. Under new environment, close associations and exposure to unusual climatic conditions, such organisms take on new life and vitality and bring about disease of the middle ear and subsequent abscess of the mastoid process. The ordinary pathogenic organisms are the three forms of streptococci and the *Staphylococcus pyogenes aureus, albus* and *citreus*.

CAUSES OF COMPLICATIONS

Any foreign substance that prevents perfect drainage is a productive cause of deeper complications and continued infection. The least deformity of a healed wound and a quick recovery is

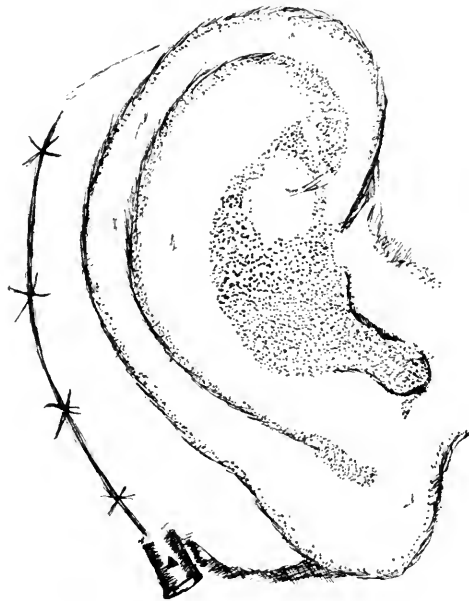


Fig. 2.—A straight tube leading to the antrum, the most dependent portion of the posterior wound. In this case irrigation of the wound is easily performed. In the case represented, to prevent the tube from falling out of the wound a ten-day catgut suture is passed through the tube and the lips of the wound. As soon as the wound becomes sterile all drainage may be stopped.

"le grand essentiel" in a mastoidectomy, whether simple or radical. The chief otologist in a base hospital apparently has the best opportunity that the country can afford for developing his skill as an operator.

METHODS OF TREATMENT

There has been a wonderful revolution in the treatment of wounds in consequence of the European war. Before the war it was the custom to adopt the "closed method" in treating wounds

and depend on phagocytosis to keep the wound free from infection.

In mastoiditis we have an infected cavity and no external operation can make it sterile. It has been the custom for many years to pack the wound with gauze, sometimes lightly and sometimes tightly. Then came the blood clot operation and its many complications. The habit of packing and the bad effect of the treatment of the mastoid wound was more particularly impressed on me years ago in Heine's clinic in Munich. The number of old postauricular openings were so numerous as to attract my attention. The habit of packing a mastoid cavity and leaving the wound to fill with granulations is not uncommon today and apparently among successful operators.

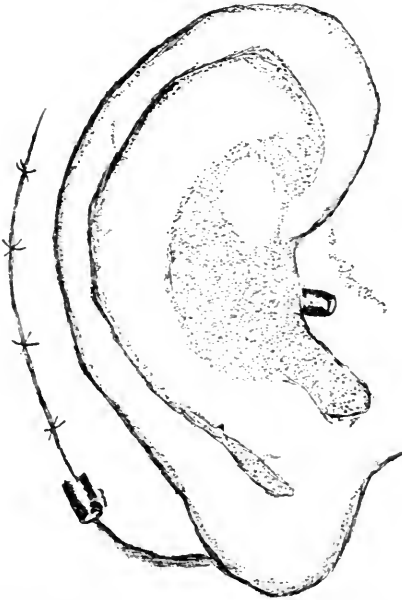


Fig. 3.—The tube passing from the auditory canal and protruding at the dependent portion of the wound in a radical operation. In some cases the tube should extend through the deeper parts and the posterior wound closed.

The usual method of packing sinuses of the head is fast becoming obsolete. Some advantages may result from the use of gauze in packing wounds of the head, but these are few and far between.

The fact that infected bone cavities have received in war surgery perfect drainage and irrigation has convinced me that the same principle may be used in mastoid cavities following exenteration of the cell. The accompanying illustrations show

one method at least, and there are many other methods that enable the operator to drain and irrigate the cavity in acute cases and after a radical operation on the mastoid.

COMPLICATIONS AND THEIR TREATMENT

Dread of hemorrhage was the cause of the older otologists advocating the packing the mastoid cavity. It is demonstrated that hemorrhage is very limited after a mastoidectomy as shown by the blood clot operation. For, if hemorrhage was pronounced,

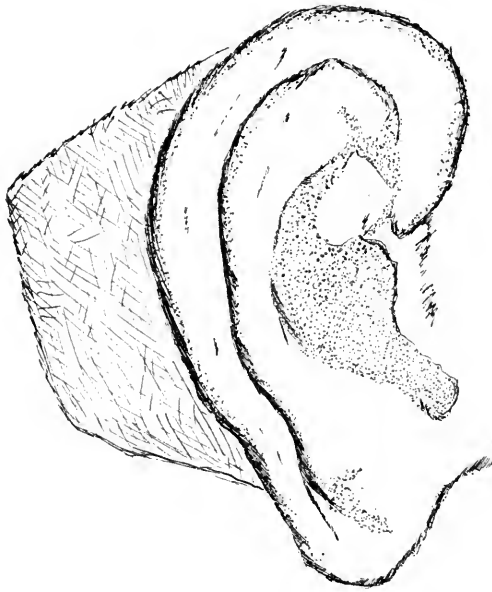


Fig. 4.—It is advisable in acute mastoid operations to avoid any dressings over the anterior part of the pinna. A small drain may be placed in the auditory canal. The dressing in the figure is indicated for the first twenty-four hours, and after that the gauze cover of the concha may be discarded.

in the blood clot operation the blood would flow so freely as to rupture the mouth of the wound and cause a severe hemorrhage.

In a serious case, such as a streptococcus infection and an infected blood stream, it is deemed advisable to fix an end to end drainage tube in the upper angle of the wound. In a radical operation a tube may extend through the wound and protrude through the external meatus.

For irrigation there are many solutions which may be used, such as a physiologic sodium chlorid solution. The solution used should always be alkaline and nonirritating to the skin.

AFTER-TREATMENT

As soon as possible all bandages should be removed. If healing is slow, the wound should be exposed to the air and sunlight. If possible, a bandage should not be used to bind down the wound, but should be held in place by a collodion dressing. Not infrequently bandages are defective and form no protection to the wound. For years I have objected to shaving off the hair of patient's head preliminary to a mastoid operation. The hair is always protected by a collodion dressing after thoroughly cleansing of the scalp with soap and water, and applying the usual tincture of iodine.

CONCLUSIONS

1. The middle ear becomes dry, as a rule, within twenty-four hours after operation.
2. The posterior bone cavity becomes sterile a few days after operation.
3. Healing is more rapid by the method described than by any other form of dressing.
4. In exposure of the lateral sinuses and dura, this method is the most logical and satisfactory way of draining.
5. An abscess in a mastoid process, ethmoid sinuses or antrum of Highmore should, as far as possible, be drained constantly, and aerated and flushed with warm salt water solution preferably twice a day. The result of long suppuration and slow healing is usually due to packing with gauze and lack of aeration.

DISCUSSION

DR. HAL FOSTER, Kansas City, Mo.: I will not detain you very long. I did not know I was to take part in this discussion until this morning.

I have enjoyed Dr. Kyle's paper very much indeed. It takes us back and reminds us of the improvement in the operation on the mastoid. I was trained in New York, at the old Manhattan, and down Thirteenth street under Dr. Buck, who had wonderful results in those days. He just simply bored a hole down in there and washed it out. I have seen him have splendid results. No doubt my friend from New York here remembers Dr. Buck and Dr. St. John Roosa, and a number of those old men, who are dead and gone now, but they had good results with their mastoid operations.

There was a man named William Barth, who came here from Kansas City years ago. He owned a building down here, I saw his name on the building today. I happened to know some of his family and I have heard him tell how he got to Denver and how Denver was named. He said he came in a sixteen-ox wagon, and a man named Denver came in his train, came the same way. I came in a Pullman. Everything has improved since those days.

I agree with Dr. Kyle in all he has said. I have never tried the open method, the sunlight method, as he speaks of it, but I believe I will try it.

I think we owe a great deal in this country to Dr. Whiting, and I have been in the habit of doing it somewhat on that style. I have also tried the method of Dr. Bryant of Boston, now of New York, with a blood clot, but did not use it very long. I think we should be very careful not to destroy the periosteum. Your wounds will heal quicker. I have seen Dr. Jansen of Berlin, and other men, who did not seem to care much how they chopped through it, with chisels and other things, but I like to lay it back and save as much of the periosteum as I can, and I find my wounds heal up very much quicker and I have very much better success.

I have enjoyed Dr. Kyle's paper very much indeed. I know he had a great deal of experience during the Spanish-American war with these mastoids. In my part of the country I do not see as many mastoids as I did some ten years ago, and in talking with the other otologists I find they have about the same experience, and I am rather glad of it. I find that the general practitioners are treating these cases better, trying to prevent mastoids, and they are preventing mastoid cases. They seem to be more wide awake to the necessity of treating young children and others with affected ears before they reach the otologist's hands. For that reason we do not see quite as many as we did.

I agree with all Dr. Kyle has said. I have never tried the open method, but I will do so the first opportunity.

DR. JOHN H. FOSTER, Houston, Texas: I have been doing mastoid work for a good many years and have tried almost every line of treatment that has been suggested. I think that in it, as in nasal work, we have all come to do away with packing more and more. Personally, I have not for a number of years packed a nose or a mastoid after operation, that is a simple mastoid for acute trouble. Contrary to the experience of many I have found the best and quickest results are obtained by following the modified blood-clot method. After cleaning out the mastoid thoroughly I close it up, except at the extreme lower angle and there introduce, not a tube or gauze, but a thin rubber tissue drain. This is left in two or three days and a large percentage of cases heal primarily. I have quite a series of cases in which the clot broke down and drainage had to be continued for some time, but even in these cases healing is remarkably short if pressure is used to obliterate the cavity and cause the scalp to adhere to the depth of the wound. Unless this is done healing is much longer delayed.

I disagree with Dr. Kyle to this extent—I do not believe in irrigating a mastoid wound. Where healing is prompt there is no occasion for it and when the clot breaks down and a large amount of discharge appears I think I have gotten better results by simply wiping the wound out with dry cotton and applying pressure than by the use of irrigations.

In radical operations for chronic suppuration I do pack immediately following the operation for the reason that I use primary skin grafts as a routine procedure in these cases. In five or six days the packing is removed and the wound treated by the open method, using simple cleansing followed by alcohol until healing is complete. Healing is quicker and the hearing better than that I formerly obtained by the use of continued packing.

DR. WILLIAM H. MERRILL, Lawrence, Mass.: It does seem as though an operation which has been under discussion for so many years should have attained a more uniform method of treatment, but after all it is very probable that we are simply treating each case in something of an individual manner. Dr. John Foster, in his discussion, called atten-

tion to the different kinds of infection. This seems to me to be very important. The hemolytic streptococcus infection is almost impossible to eradicate by the operation; we do not know whether we have got it out of the mastoid cells or even out of the solid bone, and if such wound is sewed up by the blood-clot method, or is treated by immediate closure, you are quite likely to have to do a second operation of it. If the wound is left wide open there is no doubt that the gauze should be changed very frequently, twice a day at least. It has seemed to me as though some method of rubber tubes, as we used years ago when we first began to do these things, with irrigation with the Dakin-Carrel solution, might be very helpful in these cases, where the tube is put in according to Dr. Kyle, and irrigation could be used. I think it offers something. Dr. Kyle referred to the Heath operation and I did not just get what Dr. Kyle said. There was a report, after Heath had gone back to England, the Mosher report; are you familiar with it?

DR. KYLE: I do not recall it.

DR. MERRILL: There were twelve cases which Dr. Heath operated on, and a year later Dr. Mosher, now Colonel Mosher, and I think Dr. Green, made a report on them, wholly unsatisfactory; there were only one or two cases that did not come to operation afterward. It has been my personal experience to operate on two persons who had the Heath operation performed, not by Heath himself, but by some one else.

DR. JOSEPH C. BECK, Chicago: Dr. Kyle a few years ago advocated the open air treatment for mastoid. At that time it appealed to me as a good thing, because the bandage prevented the air entering into a cavity, and in its infection certain putrefaction was aided by this tight bandage. Therefore, it is our practice to put on the first dressing, then the next morning after the operation the bandage is taken off and a wire mask is put on. This is nothing else than the mask that is used for making an ether cone, and it has straps which go around the forehead and under the neck and back of the head to hold this mask in place, and in that way the air is allowed to get to the wound, and just a light fluff of gauze is put over the wound.

At the meeting of the Academy in New York I presented the various methods of mastoid operation, by comparing them, from my own experience. All sorts of operations were given by me. The result I presented was a closed method of mastoid operation, in which, as Dr. Foster said, a small drainage is allowed behind the incision—the incision is completely closed. I have never closed up a cavity tight, except in one case, and that was when I operated in a case of furunculosis, making a mistake for a mastoid and found a normal mastoid and of course I naturally closed it up. But in all other cases I have always left a small drain, using a thin rubber tube or a light drain.

Now the change in the treatment from the old packing treatment was startling to me, particularly, as Dr. Kyle said, the ear is dry the next day or two after, and there is very little discharge then even from the posterior wound, if you have made your opening large enough from the antrum to the mastoid cavity. The fact, however, remains that that is not true in all cases; and that is due to the fact, which I have proven to myself, and think those who will study it will agree with me, that it is the pathology that is underlying these cases—the bone pathology.

At the meeting last year I showed a series of photomicrographs and slides of cases out of my practice, in which we found the following changes in the mastoid. In the acute there are two great types—and

we need not subdivide them, they merely end in one or the other type—it is the cell route infection, that is, the cells become distended with material, just a natural mass, and the membrane and the bone are necrosed by the accumulation of this material, and one cell coalesces with the other and practically the whole mastoid is destroyed, the sinus exposed. These are the cell route infections, usually syphilitic. Usually in all rapidly healing cases the microscope shows these changes. The other is what is known as the thrombotic route infection, in which the bone is fairly well preserved, but you will find, under the microscope, that the blood vessels are blocked, and those are the cases that it takes a long time to heal and are associated frequently with sinus thrombosis and epidural abscesses. Those are the two great classes, being acute inflammatory conditions, and it does not matter whether you irrigate or do not irrigate, it will take a certain length of time for the cases to heal. Of course, we appreciate the fact that with a thorough operation the case will heal quicker than when the cells are left unopened, or if the antrum is not drained; but the fact is it is the pathology, and the pathology has to be considered with the bacteriology, as our lieutenant friend said.

There is another point that Dr. Kyle touched on in the Heath operation. We all really ought to agree on that subject, that that operation has just one indication, and that is in children, in chronic cases, that is subacute, where you may do a simple operation, or do the Heath operation if you will, if that has any advantage, but do not destroy the membrana tympani and do not touch the ossicles. I see a reason for such an operation as that. In chronic cases, chronic suppuration in the attic, where the suppuration has existed for a long time, I see no reason why we should bother with that at all. I at one time said that if some one had a bilateral chronic suppuration, and in one ear there was fairly good hearing, I should do a Heath operation on that ear.

One more point: Dr. Kyle mentioned something in reference to cantonment infection. It was interesting at the last A. M. A. meeting to hear the reports of officers from different cantonments—reports of the different cases. One from the East somewhere, I forget the name of the gentleman who reported, I think had something like 250. Practically every case came to operation. And by comparing their notes, we found they were dealing with entirely different conditions, different cases. One was a very apparent streptococcus infection, many of them had pneumonia, bronchopneumonia, and others had simply influenza, with mastoid complications.

DR. W. LIKELY SIMPSON, Memphis, Tenn.: I would like to rise to say just a word about the Heath operation. Two or three of us down our way believe in the Heath operation in certain cases. I want to say that they do not always understand the case they are talking about. Take a man with very good hearing in one ear, a small perforation discharge that you cannot stop, there is only one operation and that is the Heath operation, and it will stop it in nearly every case. It has a great deal better chance of stopping it than an ordinary simple mastoid. The Heath operation gives a permanent drainage there that you cannot possibly get with a simple mastoid, and that is the very reason that it is better.

DR. JOHN J. KYLE, Los Angeles: I want to thank the men who have taken part in this discussion. I think if you will try this open drain, and do away with packing and bandages, you will be very well satisfied. The recovery is so quick. I had a boy who came into the hospital a short time ago with a double sinus thrombosis and in about five days

he was out of danger, and in about three weeks he was absolutely well, and that because he had perfect cleansing. Bandages cause a great deal of pain. After this I am going to adopt Beck's method and put on one of those little masks, which is the quickest way out of it.

As regards the Heath operation, I have performed that many times. I remember Ballenger advocated that operation, and I was very much amused, up at this congress, we were all there, Beck, Stein and myself, and Heath had a method of getting his hands back this way (illustrating) and running around like a kitten, and one of these Englishmen arose and took much exception to his remarks. But after all, the operation is quite a success.

The whole thing in a mastoid and middle ear is to have no back pressure in the mastoid cells because there is a constant flow of pus through the middle ear.

DR. FREDERICK STAUFFER, Salt Lake City: I think the Heath operation is misunderstood by a large majority. For instance, Ballenger, in his book, describes the Heath operation, and it is not the Heath operation at all. I worked with Heath quite a long time, and I know that he has very fine results in his operations. I think the great mistake Heath makes in his operation is that he does not take out all the mastoid cells, as is essential if it is to take the place of a radical operation.

His operation was devised for the purpose of substituting the radical operation in selected cases. He calls it a modified radical. I think where Heath fails mostly is that he only takes out the cells of the antrum, of the mastoid instead obliterating all the mastoid cells. But if his operation is done properly—in children, for instance, you take out all the mastoid cells, and pay a good deal of attention to his technic down in the antrum you are going to get fine results. A house surgeon that had been there with Heath for a year said that Heath's cases recovered much more quickly and had a dry ear much quicker, than the cases of any other men that operate in Golden Square Hospital. I think it is a distinct advantage for operating on children, that is, by making Heath's operation around the tympanic membrane, taking all the cells—do not leave any of the cells, as I have seen him do in adults; but with a flap closed up absolutely, there is no scar left, and the ears are dry in a very short time. He does not use any packing at all, he uses a rubber tube to keep the flap in place, and he uses glycerin emulsion of iodoform as a dressing, and it is astonishing how quickly those cases heal up.

DR. BECK: How long has Dr. Stauffer seen the cases after operation?

DR. STAUFFER: About three or four months, some of them. But what I was saying was that the man who was house surgeon for a year said that his results were very good. I did not see any of his cases that long afterward; but the immediate result was certainly good.

SPEECH-READING AND ITS VALUE

BESSIE LEWIS WHITAKER

DENVER

The honor of speaking to this Academy on the subject of speech-reading is one of which I am deeply sensible.

In the office of the otologist — sometimes perhaps even in the oculist's office — the hard-of-hearing person not only finds that abnormal physiologic processes tending to blight life may be warded off, controlled and checked, but there he learns also that there is still another avenue of constantly increasing relief open to him — the way of lip-reading. Through a nice adjustment and training of the eye and the coincident bringing into play of the powers of the brain, he may actually understand the spoken word through the channel of eye instead of ear communication. This specially acquired type of seeing power may either supplement or take the place of hearing power. The degree to which the ear is used in cooperation with the eye is represented by the exact amount of hearing power possessed by any particular individual. The far-reaching effects of lip-reading power on the life of the hard-of-hearing are of a personal, social, economic and moral nature.

SPEECH-READING ADAPTED FOR THOSE SLIGHTLY DEAF AS WELL AS FOR THE TOTALLY DEAF

Who should study lip-reading? Dr. Jackson has most kindly suggested that you might like my answer to this question. There is value in lip-reading even for those whose hearing is not impaired. We echo Dr. Clarence Blake's prophecy just from the press that general acquaintance with speech-reading will ultimately lead to its inclusion in the stated curricula of our schools and colleges. At the present time we are realizing as never before the need of help for the hard-of-hearing. The slightly deaf person, missing in conversation only a few words here and there, cannot resort too soon to the aid that comes through speech-reading. The totally deaf, even though brought suddenly by some war injury or other calamity to a soundless world, may return to a plane of understanding the spoken word by means of the eye, and doing this means the return to normal life. The

deaf child of three years may begin the acquirement of this eye power through being led to watch his mother's mouth as she speaks to him. Undoubtedly little hard-of-hearing children, struggling pathetically with their school work under the handicap of deafness—whether it be of slight or great degree—should be rescued through technical training in speech-reading. Boys and girls of high school age, suffering keenly from the condition of mind which the deaf know even when deafness is in an early stage, should be rescued likewise. Within the last fifteen or twenty years, as you know, a special campaign has been instituted throughout the United States for the saving of the adult hard-of-hearing through methods of teaching definitely adapted to this class, including the youth who have already learned to speak normally. As now widely recognized, these persons with partial hearing and full speaking power need instruction of a very different nature from that required by the congenitally deaf—the class of deaf we once called deaf and dumb—whose interests are carefully safeguarded in the many splendid institutions throughout the country.

SPEECH-READING FOR DEAFENED SOLDIERS

At this time when our feelings are so bound up in the questions pertaining to bringing back our injured soldiers to normal life, we are realizing the greatness of the change in life prospect that may be brought about for the man deafened in war through giving him the power of speech-reading. Until the government is ready to muster its already organized forces for this work of rehabilitation, our school in Denver stands ready to serve. Physicians are requested to tell us when this tuition should be given entirely free of charge. The request is specifically explained in the folder containing statistics from the school which I have taken the liberty of submitting to you—in response to a most valued idea of Dr. Carmody's that you might care to know something of our work in this city.

STUDY OF SPEECH-READING SHOULD BE PRACTICALLY UNIVERSAL

If every third person between 20 and 50 years of age is more or less deaf in one ear, according to the estimate of Dr. Harold Hayes, if the conservative estimate of the number of hard-of-hearing in New York City alone was placed some time ago at 100,000, it would seem that the study of speech-reading should

be practically universal. It is my opinion that all who have no serious defect of vision (barring only the feeble-minded) can become speech readers, provided the prescribed practice of technical exercises be carried out absolutely. Becoming a speech-reader, however, does not mean becoming a perfect speech-reader. As long as speech-reading is an art and not a science there is no such place attainable. Success is in different degree for each individual. Failures may at times discourage, but I believe the general life benefit is unmistakable in all cases.

EXTENT OF THE STUDY OF SPEECH-READING

What proportion of the heard-of-hearing do study speech-reading? I have been unable to find any figures in this connection in the columns of the last census report. But the discussion of lip-reading in the report for 1900 is interesting. Approximate figures in round numbers are as follows: 90,000, deaf; 15,000, lip-readers; 14,000, non-lip-readers; 61,000, ability not stated. The inference is that the 61,000 do not read lips; that speech-reading power does not belong to the partially deaf and the fact is accounted for in artificial conditions not inherent in the nature of the case. The hard-of-hearing turn the head for use of the best ear and consequently do not look at the mouth. Persons speaking to the deaf shout in the ear, thus keeping the mouth out of view. Many of the trumpets require placing the mouth out of view. Obviously the first requisite for attaining speech-reading power is watching the mouth.

DEFINITION OF SPEECH-READING

The popular definition of speech-reading as hearing with the eyes can hardly be surpassed for clearness. The speech-reader with partial hearing does not know how much he sees and how much he hears. He does not think of lip movements, tongue movements, cheek movements, throat movements, and certainly has no interest in the invisible formative movements. The power of quick recognition of visible movements in following speech — in many cases acquired only as the reward of persistent, purposive, well defined, well directed practice in watching speech through series after series of especially designed exercises — has with the lip-reader become subconscious. He does not think of the movements he sees any more than you think of the analysis of the sounds in the words and sentences you hear. Speech-reading is the interpretation of speech through terms of the visual

instead of the auditory. As most aptly described by Dr. Clarence Blake of Boston, it is translating speech by sight.

Is the blind person to be enabled by means of the new phon-opticon devised by Dr. F. C. Brown of Iowa to read print in terms of sound? Is the little box called the eye, running over the line of print, to change light waves from the letters of the print into sound waves? Is the blind person, hearing through a telephone receiver musical sounds made by an electric current, to be able simply to follow the thought conveyed by the printed page in terms of sound? If so, the *blind see* in terms of sound. The lip-reading deaf, without the aid of any artificial box, make of the eye and the brain an "ear" as sensitive as any crystals in the blind person's "eye box." The deaf hear in terms of sight and without the aid of any artificial medium when they simply see what you say.

SENSITIVE SIGHT IMPRESSIONS NECESSARY

The eye and brain of the lip-reader must be, as it were, very sensitive to sight impressions. My first desire is to adjust a physical factor by sending a new pupil to the oculist — not for "lip-reading glasses," though I wish indeed we could obtain these — but because we should be able to assume that his eyes are in adjustment for general vision. If this is the case there is in my opinion no occasion for eye strain, no excuse for it if the pupil rests his eyes frequently during the time he is learning to observe closely. I saw my nose when I began the study of lip-reading in the Müller-Walle School in Boston, but was relieved as soon as I forgot to cross my eyes in the distorted intensity of gaze I had fancied necessary.

METHODS EMPLOYED

The time limit for this talk prevents my giving even in rough outline the scheme of the Müller-Walle and Nitchie methods. As you are probably familiar with the distinguishing features, I shall merely state that the choice of methods in our school means only the pupil's initial choice of material. Mr. Nitchie — a great teacher and expert lip-reader, a personality sadly missed throughout the whole country since his death in October, 1917 — told me he considered a teacher better equipped for having had the Müller-Walle normal course in addition to his own. The principle inculcated in both schools is the study of the pupil for his individual needs. The mode of description of visible sounds is not quite the same in the two schools; the plan of building up

the exercises is different; the method of handling narrative material varies. In the Nitchie as well as the Müller-Walle school, sounds were studied as movements in ever varying combinations, not as fixed positions. In the Müller-Walle course there is abundant opportunity for developing those qualities of mind so accurately, systematically and methodically developed by Mr. Nitchie as perhaps his foremost contribution to the cause of speech-reading. These qualities of synthetic power, intuition, quickness, alertness and the power of concentration are brought about coincidentally with the training of the eye for accuracy, quickness, visual memory and subconscious power.

PSYCHIC STATUS OF THE DEAF

The hard-of-hearing are unquestionably morbid until the evil spell of deafness is cast off by lip-reading. The subjective aspects of deafness as analyzed in an article of great force by Dr. George W. Johnston are shyness, petulance, consciousness of imposing inconvenience on others. In a few cases I believe the shyness displaced by aggressive distrust. As the author referred to states, the deeper lesions of deafness are of character and disposition and the problem is to find a possibility of "achieving character all the stronger, life all the happier, success all the sweeter" because of the very difficulties encountered. If this is to be done it must be along the path of speech-reading. The late Dr. D. B. St. John Roosa was evidently interested in the subjective side of deafness when he urged his patients to learn to avoid the shrinking from human contact. I well remember his encouragement for the keeping up of my work at a time when realization of deafness was new to me. The fact is—a state of being hard of hearing in connection with some of the attendant ills, is something like being under a sentence of solitary life imprisonment under peculiarly hard conditions. To account for the proverbial cheerfulness of the blind and the moroseness of the deaf—or if it be more accurate to so state their comparative positions, the resignation of the blind and the resentfulness of the deaf—it must, of course, be remembered that horrible as is the loss of vision, the sense of hearing left to the blind is a channel of communication with other human beings.

SOCIAL AVERSION COMMON AMONG DEAF PATIENTS

As this oral means of communication is lost, either through gradually growing deafness or sudden deafness, there is lost to

an extent the bond of human sympathy. We do not like to mingle with others merely to be made conscious at every point of the impossibility of enjoyment of intercourse. Moreover, we want no pity. And often an innocent smile we fancy at our expense is a sword thrust. I verily believe that many of the hard-of-hearing would like to hide in caves. I have had the impulse myself. The consciousness of being shut out from the possibility of usefulness is keen suffering. It may lead to bitterness that has a far-reaching and sometimes even irremedial effect on character. We feel that ambition must be crushed. Even the desire for education must be stifled. The iron chains of deafness hold us down. Avenues of personal improvement, social pleasure, business or professional advancement are seemingly closed.

SPEECH-READING ALWAYS GOOD ENOUGH TO WIDEN SOCIAL
RELATIONS* AND PROFESSIONAL OPPORTUNITY

But the power of speech-reading tends gradually to bring about adaptability to environment. The deaf lip-reader ceases to be a recluse. Interest in the interests of others grows through the power of oral communication with others. Self consciousness may even disappear. I am told at times that health has improved through the relief from nerve strain that comes with reading the lips and even that actual hearing is better for the same reason.

The hard-of-hearing are finding more and more that speech-reading power is an open sesame to many commercial positions, that they need no longer remain in the cave of unhappiness, of no avail in the affairs of men. There is work in the world for the speech-reader.

DISCUSSION

LIEUT.-COL. CASEY WOOD, Chicago: Regarding some of these injuries, one almost celebrated case came home from the Front, went through several hospitals, was examined by a number of ophthalmic surgeons, but none seemed to recognize the hysterical character of the symptoms. Finally, he fell into the hands of an osteopath who, as soon as he "examined" him, recognized the condition as "dislocation of the second cervical vertebra," and said "I can cure you in five minutes." He reset the dislocated (*sic*) vertebra, and sure enough a speedy cure followed, and he is now to all intent and purpose just as good as ever! One cannot blame the discharged soldier and his family for believing this to be an instance of blindness from injury that was cured by his osteopath doctor "when others failed."

DR. H. G. MERRILL, Provo, Utah: One can see readily how difficult it would be to read the speech of the very careless speakers, such as the average Americans. This care in proper enunciation has received some

* Expression "To widen social relations," quoted from Dr. H. B. Young of Burlington, Iowa.

notice in the East, and Dr. Swift, in Boston, has a school and some teachers in the public schools, and in Philadelphia is such a school. This is a movement in which each individual can assist, and a movement in which the public schools can be of greatest help.

Colonel Wood alluded to a notorious case of osteopathic amblyopia. The important part was as to how this particular case was imposed on one of our own associates and used to collect money. This is even more curious than the failure of the medical profession to recognize the character of the injury.

DR. H. B. YOUNG, Burlington, Iowa: While in no way wishing to detract from the things accomplished in the rehabilitation of the deaf, I would call particular attention to a preliminary statement by the first essayist, that "Speech reading is an art; and some naturally get it better than others." This is a fundamental truth not mentioned sufficiently often. Nor is very much said of two essential factors in getting this art, namely, mental alertness, and quickness, with accuracy of vision above the average. There has, of course, been a clamor for it ever since Helen Keller was exhibited to the world, due mostly to the too easy conclusion that what had been done for one both deaf and blind should be possible for all the deaf not blind. But real artists, after all, are few; and the other kind get little consideration outside of the ever narrowing circle of intimate friends. Moreover, the lesson to be learned in the "follow up" of the most promising pupils is not sufficiently regarded. It shows that the majority revert to the society of the deaf where the sign language is used infinitely more than speech. When I brought this subject to the Academy's attention, four years ago, in Boston, I had a lady demonstrate the Lord's Prayer in the sign language. It was a surprise to know that she was one of the best lip-readers in the country; so fine that she had been used by Alexander Graham Bell to demonstrate his pet hobby. Last year in Hartford at the centennial of the founding, by Thomas Hopkins Galaudet, of the first American school for the deaf, where I had the honor of a place on the program, I was greeted by a lady that I did not at first recognize, but who quickly identified herself as my Boston assistant; and as she could read my lips a pleasant acquaintance was renewed. On all sides were deaf people talking with their hands, and it was noteworthy that my lip-reading friend was no stranger to many of them, as she turned to hail one after another in the sign language. Among the highly educated deaf whom I personally know there are: Hodgson, collegiate M.A., editor *Deaf-Mutes Journal*, author of some verse that will fit in Tennyson's "In Memoriam," speaks well (read his address in Hartford), but confessedly a poor lip-reader; Cloud, collegiate M. A. and D.D. (addressed this Academy in Memphis) is poor lip-reader; Bayless and wife, teachers in Belleville, Ontario, and others. Bayless and Mrs. Bayless, however, will best illustrate my contention. Mr. B. lost his hearing when 17; Mrs. B. at 7 years of age. Mr. B. speaks well (of course); Mrs. B. just passably. Mr. B. cannot read lips at all; Mrs. B. is a premier lip-reader. It is unthinkable that with such advantages Mr. B. should have made no real effort. Speech reading not good enough to widen social relations therefore spells *tyro*. Nevertheless, if I had a deaf child I would have it learn speech reading if it could. But first I would myself learn the sign language (the only universal language) so that I would be on its level and be a real companion in building up. If our soldiers in France had had the rudiments of the sign language what an immense advantage it would be.

DEMONSTRATION OF NEW INSTRUMENTS FOR ENDO-
BRONCHIAL TREATMENT OF ASTHMA; MICRO-
SCOPIC SPECIMENS OF TERATOMATOUS
GROWTH OF THE TRACHEA

WOLF FREUDENTHAL, M.D.

DR. WOLFF FREUDENTHAL, New York City: If you believe as I do, that bronchial asthma is essentially a disease of the bronchi, it is our duty as laryngologists and bronchiologists to treat the bronchi directly, that is, endobronchially. For that reason I have been employing the method advocated first by Dr. Epstein of Breslau, that is, with his endobronchial spray, a very simple method, indeed. I modified it so that it can be used in any office by connecting it with the compressed air apparatus. We first used adrenalin alone, but I very soon saw that we did not make much headway, and I applied astringents afterward. Then there is undoubtedly in some cases an atony of the bronchial musculature, and that can only be overcome, according to my present view, by applying the galvanic and faradic current endobronchially. All this can be done through this spray when connected with a battery. I have it here connected with my spray. It can be used in the office to great advantage. After introducing the tube to the right or left side, I spray in first cocain or adrenalin, or both. Then the electric current can be applied through the same tube and finally other medication used. Since the war broke out, unfortunately, we cannot get this tube, nor could an instrument maker be found here who would make it at the present time. The introduction of this tube is very simple. You take hold of it this way (illustrating), and under the guidance of your mirror you go in between the vocal cords holding the end of the tube by means of a Krause forceps somewhat modified by me. The further introduction is easy.

The results are so far encouraging. I must admit I do not have 100 per cent. cures, nor 90, but I believe that this is a field in which we should work. One of the causes of my failures is that I am still afraid to use stronger astringents. Adhesions may be formed further down as it happened in the nose, and for that reason I am careful.

LATE APPEARANCE OF FACIAL PARALYSIS FOLLOWING MASTOID OPERATION

HARRY A. SMITH, M.D.

DELTA, COLO.

There is, in the whole category of surgical experience, no accident more distressing and humiliating, from the point of view either of the doctor or of the patient, than the deformity incident to facial paralysis, and a lively appreciation of this fact has exercised a wholesome restraining influence on the ardor of many an inexperienced, enthusiastic disciple of otology who would otherwise have been disposed to assume unwarrantable risks.

To the beginner in the surgery of the ear the mere suggestion of paralyzing the face is an appalling prospect, while even the seasoned campaigner cannot contemplate such a catastrophe with any degree of equanimity; as for the sensations of the patient, it is useless to attempt to analyze them—no language can adequately express the combined mortification and disgust which the staring eye and distorted visage daily and hourly inflicts on the hapless victim of this unfortunate accident, a distress which the surgeon is by no means always justified in attempting to mitigate by the assertion that the deformity is but temporary.

TIME OF APPEARANCE

Before this audience, no review of the technic or dangers of the simple or radical mastoid operation are necessary. It has been noted by Mahn "that a facial paralysis after operation is not necessarily due to injury of the nerve." It is this fact that I wish to call attention to. In a case that has been operated on, in which there has been no operative complication at the time of operation and apparently a smooth recovery began, it is with much chagrin to the operator and annoyance to the patient that a facial paralysis begins to develop from as late as the ninth to the sixteenth day. The many factors that might give rise to a paralysis have been considered, but none of them account for the excruciating pain over the distribution of the facial nerve. The

seventh nerve is credited only with the motor function and, in the two cases to follow, as much care has been taken as can reasonably be done to discover the origin of the pain, and it cannot extracranially be accredited to the fifth nerve.

ETIOLOGY

It may not be out of place to mention a few of the causes of facial paralysis. Contusion of the nerve without laceration; compression of the nerve; inflammation; exposure to severe colds; dehiscence of the bone with retention in the middle ear causing compression; localized meningitis; and fracture of the petrous portion of the temporal bone. But none of these causative factors account for the following conditions.

REPORT OF CASES

CASE 1.—Miss B., aged 30, has been under observation for five years. She came from Luxemburg. Following scarlet fever at 8 years of age, both ears discharged. She was treated in France, Germany and Luxemburg. In 1915, by use of autogenous vaccine, both ears stopped discharging. In 1916, the left ear began again to discharge. In June, 1917, a radical operation was done on the left ear, at the Mayo Hospital, Rochester, Minn. On the ninth day after the operation a complete facial paralysis developed. The patient returned home, and again came under my observation about August 1. The paralysis at this time was complete and the pain intense. By the end of the year 1917, the paralysis had almost disappeared. There is still a very slight evidence of it about the eyelid, but the pain continues. Since the recovery of the facial paralysis and the continuance of the pain, a Wassermann test has been done, a roentgenogram has been taken of the teeth and mastoid process and nothing can be found to account for the condition.

CASE 2.—Mr. M., aged 36, was an American, single and of good physique. The patient gave a history of discharge from the ear for twenty years following scarlet fever. There was a foul discharge from the ear, hearing was practically nil, the posterior wall was sagging and almost filled the canal, and there was tenderness over the mastoid region. The patient was operated on by the writer July 1. The entire mastoid region was eburnized. The operation was without event except that the deeper part of the mastoid was very hard. He was returned to the bed in good condition except that the pulse was rather slow, and continued slow for six days — from 50 to 60. The temperature arose on the third day after the operation to 103 F. for a few hours then dropped back, and at no time after this was it higher than 99. Only July 15, or fifteen days after the operation, he complained of severe pain and on the next day facial paralysis

began to develop, which in the next three or four days became almost complete. In a few days he began to improve and at this time the paralysis has almost disappeared, but the pain some days is as intense as it has been at any time, necessitating the giving of drugs for its control.

DISCUSSION

DR. WILLIAM C. BANE, Denver: I do not think I can add anything to the doctor's paper. It is interesting to me. He is to be congratulated on the results, the rapid recovery in the two cases. It has been my lot to have one case of facial paralysis showing up after radical mastoid operation, much to my chagrin. I was not aware at the time that I had done any damage to the nerve. Just what occurred I do not know. The nerve was injured but not destroyed, for within three months the functions were fully restored. I have nothing further to add.

DR. W. V. MULLIN, Colorado Springs, Colo.: What the doctor has said in his paper, recalled to me a case that I am observing at this time. Last Wednesday afternoon, I did a radical mastoid operation on a girl 28 years old. It was a pure influenza infection of long standing. Immediately following the operation, there was no paralysis. I watched for it very carefully. The bone was dense and eburnated and was very difficult to chisel away at the facial ridge. There was some twitching of the face during this manipulation. I was impressed on reading a few days before, in the *Laryngoscope*, a report of Dr. Philip Kerrison of New York, for using dichloramin-T after the radical operation, and I thought I would try it. I used it immediately after the operation for washing out the cavity, and I instructed the nurse to drop in the ear a $\frac{1}{2}$ of 1 per cent. solution every hour. This was done for four days. On the evening of the fourth day the patient had more pain than usual. This fact was conspicuous because she had had very little pain following the operation. The following morning there was a facial paralysis on the left side. It was not complete as she could draw back the angle of the mouth a little and could partially shut the eye. I discontinued the use of the dichloramin-T as I was of the opinion that it was producing some irritation and affected the nerve. I just heard over the telephone, less than an hour ago, that the patient is all right today.

One other case I had, was in a boy 20 years old. This was a tuberculous ear, demonstrated by pathologic examination and injection into a guinea-pig. On the tenth day following the radical operation, I had a complete facial paralysis which lasted for four days and then gradually disappeared. Strange as it may seem, this boy has since been accepted into the Army, and I had a card from him from France the other day. He had perfect healing. In this case there was a great deal of softening of the bone and the rapid clearing up of the paralysis pleased me very much.

DR. FREDERICK STAUFFER, Salt Lake City: I have been led to believe that a good many of these cases of paralysis came on after operation, when the nerve itself is not injured, from back pressure of the secretions when a packing has been used. I think that is about the only time I have seen it. I believe that by paying more attention to the kind of solutions we use, or not use them at all, we will have less facial paralysis.

DR. JOSEPH C. BECK, Chicago: The last two speakers brought out the point that I wish to make, and I have had experience with facial paralysis, have seen cases after operation, and both of the cases Dr.

Smith reports would lead me to believe that those were traumatic irritation—paralysis from manipulation. I have seen that, during my service in the Cook County Hospital. The radical mastoid operation has exposed the bone in the region of the facial nerve too close for any man who is not qualified to do special work, and in public hospitals we must depend oftentimes on insufficiently trained men, so that in manipulation such a thing may happen, and it may often happen to a trained man, that he irritates it with a probe, traumatizes it, so that it would result in a temporary facial paralysis. I had a unique case of this kind. About two weeks after I did a radical mastoid operation I wanted to close the tube completely, so I syringed out the tympanic end of the tube, and then I took a probe which is flexible and allows you to apply a very small part of cotton on that, and I dipped that in pure carbolic acid with the idea of destroying the tympanic end of the tube.

Evidently some of the acid, or at least I believed that is what it was, although I used alcohol immediately afterward, touched the canal. Facial paralysis developed but disappeared in three or four weeks, without any after-effect. I took photographs of that patient showing complete facial paralysis.

Now the other point is this, that if a man does have complete facial paralysis, he ought not despair, the condition is repairable by a plastic operation.

DR. D. D. McHENRY, Oklahoma City: One thing that has not been brought out is the length of time that this paralysis has persisted. I had a case coming on about ten days after a radical mastoid operation—in which I used packing—which continued for three months before I got any sign of relief. I had given up all hope of getting any cure, when I gradually began to get action of the muscle and finally it entirely cleared up.

This is one thing I would like to ask Dr. Smith, and that is how long has this paralysis persisted?

DR. SMITH: The first lasted about five months and the other about five weeks.

DR. McHENRY: Another case that came under my observation was that of a dentist, who was in an automobile accident. He had without any question a fracture of the base of the skull, and we had all given up hope of any relief from the facial paralysis, thinking that the nerve had been cut off by the fracture. Nearly five months after it gradually began to come back. Three months later it was entirely recovered or eight months after the accident.

DR. S. H. LARGE, Cleveland: I want to state a case, because I want some help on it. I performed a radical mastoid six months ago, on a young girl of about 18, and forty-eight hours after facial paralysis came on, quite complete. In this case there was no packing used. The facial paralysis was complete for about a month. Then it started to clear up. But the peculiar thing about the case was the pain. This girl had excruciating pain, worse at night; in fact, she had to be kept under opiates, and a peculiar thing about it is you cannot touch any part of the scar but it gives her excruciating pain. I thought perhaps it might be hysterical; but the girl is losing in weight. The facial paralysis is clearing up now, she can close the eye perfectly, but the lower part of the face is still paralyzed. There is no discharge. We have her in the hospital now, under observation, and I would like to have some suggestions as to what to do for the pain.

DR. J. J. KYLE, Los Angeles: If I had an opinion to express offhand about that pain, I would say it was an irritation of the otic ganglion,

a very hard thing to treat. It might be from the tube. I have seen that often. Now to cure an irritation of the otic ganglion is certainly a hard thing, but I would think the roentgen ray applied once in a while, not too long, would probably be the best thing you could do. I know that I had a traumatism in my leg followed by a terrible pain so that I could not sleep at night, and that the roentgen ray was just marvelous—it stopped the pain absolutely. It seems the best thing to do.

DR. WILLIAM H. MERRILL, Lawrence, Mass.: Let me repeat that most of these cases do get well, and let me call attention to the work at the Johns Hopkins Hospital. More than 75 per cent. of the patients taken there showed trouble in their middle ears. They had had a subacute infection which had produced paralysis of the facial nerve. When these unfortunate things have happened in my practice I have thought it was not always trauma, but an infection.

DR. R. P. SCHOLZ, St. Louis: I would like to tell of a case that I had, together with Dr. Kaplan of St. Louis. A child, about 5 years old, had had acute otitis media of several days' duration, and suddenly a facial paralysis set in. The child was operated on the following day, and the paralysis persisted for about three months, and then cleared up gradually. But since then, and that has been now about two years, there is an increase in the dimensions of that side of the face, which does not seem to be loss of tone in the muscle, but seems to be rather an increase or spread or hypertrophy.

DR. HARRY A. SMITH (closing discussion): You and I are having the same kind of trouble. They cannot go home with a paralyzed face and go to sleep, and then call us up in the middle of the night. These two patients of whom I spoke suffered excruciating pain, and I have had trouble controlling the pain. In one, operated on a year ago last June, I still have to give codein or something to control the pain; the other I have trouble with about two days a week. The pain apparently follows the distribution of the facial nerve up on the side of the eye, around there. From the pain you would think it was from the fifth nerve. In these cases roentgen rays have been taken of the teeth, and the patients still continue to have pain. Doctor, I sympathize with you.

SOME OF THE MERITS AND DEMERITS OF THE GUARDED AND UNGUARDED WIRE SNARE IN TONSILLECTOMY

WILLIAM C. BANE, M.D.

DENVER

The enucleation of a tonsil with its capsule or basal membrane without additional tissue is the goal we aim at in doing a tonsillectomy. In using the snare whether the wire is guarded as in the Beck-Schenck instrument or unguarded as in the Tyding's snare, it is necessary to examine carefully the relation of the wire to the tonsil to feel assured that only the gland will be removed. Who of us have not been surprised and chagrined to find on examining the fossa that a part of the gland or a part of the posterior pillar or even the uvula had been severed. While I have used the Sluder instrument I am not qualified from experience to comment on its merits.

PARTIAL DISSECTION METHOD

For many years I have followed the partial dissection method with scissors and knife, completing the operation with the unguarded snare. The most trying part of the dissection to me has been the freeing of the posterior pillar and the adjacent connective tissue so that no part of the posterior pillar or its mucous membrane would be drawn into the snare. Quite frequently, especially in adults, the connective tissue is dense, and unless cut by scissors or knife it will not yield readily to the snare but tear away with it the adjacent muscle that is less resisting. Once removed there is no replacing the muscular tissue to restore the muscle to its normal position.

GUARDING RING

The guarding ring may be used after partial dissection, the tonsil being drawn through the ring that is held firm between the posterior pillar and tonsil, thus preventing the pillar from being drawn into the snare. The ring selected should be as small as will just permit of the tonsil being forced through it. In the use of the Beck-Schenck snare, or similar instrument, the ring is placed under and posterior to the tonsil between the gland and the pillar and raised while with the forefinger of the other hand

against the anterior pillar the tonsil is pressed through the ring and held there while the wire enclosed in the ring is tightened on the tonsil. Then the gland is removed rapidly or slowly as seems desirable. The wire completes the dissection so that it is essential that the gland be grasped with forceps before the operation is completed.

POSSIBLE ERRORS IN TECHNIC

Recently in tightening the wire on the tonsil in a 3-year-old child the wire cut completely through and the tonsil dropped out of sight. Fortunately I was able to bring it into view with the finger while the assistant secured it with forceps.

Occasionally with the guarded snare there occurs the loss of a narrow strip of mucous membrane, but none of the muscle. If all of the gland has not been forced through the ring a portion of it will be found in the fossa.

COMMENT

The guarded snare method like the Sluder method is not adapted to all cases, especially in adults who have had frequent attacks of tonsillitis and peritonsillar abscess, as the adhesions are so firm that they will not stretch enough to permit the forcing of the tonsil through the ring. In such cases the partial dissection with scissors and knife will serve best, completing the operation with the unguarded wire snare.

A TECHNIC THAT OBVIATES GENERAL ANESTHESIA IN THE EVAGINATION OF TONSILS

THOMAS L. HIGGINBOTHAM, M.D.
HUTCHINSON, KAN.

It seems that the day is near at hand when fetichism, sentiment and selfishness will be all that remains to justify the administration of general anesthesia when removing tonsils.

EFFICIENCY OF LOCAL ANESTHESIA

The virtue of local anesthesia needs no champion; its use being established on such a solid foundation of logic and safety that instruments and technic must conform to its demand. It is sounding the death knell to fads and fancies and stamps the luxurious display of armament and assistants as unpatriotic extravagance. It is destined to make the administration of general anesthesia for tonsil surgery a moral and legal misdemeanor.

It is generally agreed by those with discerning mechanical minds that the art of tonsil enucleation rests on the principle of "evagination," and, in all probability, offers the only remaining hope for the redemption of tonsil surgery from the mutilating hands of ignorance and conceit. It is with certainty that we say that it is the foundation on which we must erect our tower of expert individuality.

Eternal honor is due to him who first applied and popularized this principle to this field of protective surgery; it alone is sufficient to place his name in the Hall of Fame were he not crowned with other triumphs, and, in harmonizing this principle to the benefits of local anesthesia, we hope to crystallize his merited glory into diamondlike brilliancy by wresting the maneuver from the pitfalls of general anesthesia and instrumental debauchery.

TECHNIC OF ADMINISTRATION

By placing 6 c.c. of a one-half of 1 per cent. procain solution exactly into each pericapsular space pressure anesthesia is produced and acts as a hydrostatic dissector. Pressure anesthesia and hydrostatic dissection are produced by injecting 6 c.c. of a 0.5 per cent. procain solution exactly into each pericapsular space.

The patient assists by depressing the tongue as the surgeon catches the tonsil near its center with a three-tined, narrow-pointed tonsil tenaculum to make retraction medially and forward, to be incised from pole to pole, slitting the hood when phymosing, with any simple tonsil scissor or scalpel. Here the tonsil is released and the tenaculum passed through the loop of a "properly" wired lever snare to engage the tough capsule through the slit in the gland. Traction with the tenaculum causes the gland to "evert" through the properly shaped loop to be enucleated as the wire is drawn home.

SUMMARY

1. By combining the principle of "eversion" with that of "evagination" the objectionable features of general anesthesia and dissection with sharp instruments are obviated.

2. All the good features of local anesthesia and enucleation are retained.

3. The operation is as near bloodless as can be done with any dull-bladed or clamp instrument.

4. No trauma is done to soft parts by leverage or digital pressure.

5. It makes the removal of acutely inflamed and quinzied tonsils a rational surgical procedure.

6. It makes the removal of a child's tonsils no more difficult than would be the extraction of its teeth.

COMPLICATIONS AND SEQUELAE OF TONSIL AND ADENOID OPERATIONS: THEIR PREVEN- TION AND MANAGEMENT

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Although often regarded lightly by the laity and all too frequently so treated by the profession, it has been my observation that numerous complications may arise from tonsil and adenoid operations and many such references may be found in the literature. It is true that the vast majority are not fatal, and it is also probable that the actual percentage is not high, but when we come to enumerate these complications and to consider that many have doubtless never been reported, it must appeal to us that the subject is one to which a great deal of interest should attach.

HEMORRHAGE

Undoubtedly the most frequent untoward occurrence in the course of these operations is bleeding. How many cases would become serious if not interfered with it is impossible to say, but there are few men doing this work who do not feel called on very frequently to control the loss of blood. Not only does it conserve the strength and vitality of the patient, but it also diminishes the postoperative nausea by preventing the swallowing of blood.

Predisposing causes should be ruled out previous to operation. High blood pressure and arteriosclerosis must be thought of, as also should certain systemic diseases, such as the anemias, leukemias and choletrias. Hemophilia, though rare, is occasionally met with and may be easily overlooked as the subjects are usually robust and healthy looking. This mistake should be guarded against by a careful history in every case and by taking the coagulation time of the blood if there is occasion for the slightest suspicion. It is generally conceded that the coagulation time is not conclusive evidence as to whether a given case will bleed seriously or not, but it is of value in assisting us to reach a decision in questionable cases, particularly in the blood dyscrasias and cholemia. In hemophilia, the history, both familial and personal, is of paramount importance.

In dealing with this type of case following operation, mechanical means having proven useless, transfusion is the procedure of choice to combat the condition. In its stead, the use of normal serum, antitoxin, or one of the several proprietary preparations may be tried. The use of these preparations, or of emetin or pituitrin, has been advocated as a preliminary to operation in all cases, but I have been unable to convince myself that their routine use is justified or necessary, or that they really accomplish the desired result.

However, in the vast majority of cases no systemic condition will be involved, and the question is simply one of mechanical interference. There is no doubt that proper mechanical interference would have controlled most of the fatal cases of post-operative tonsil hemorrhage that have been attributed to hemophilia.

BLOOD SUPPLY OF THE TONSILS

Although a detailed study of the anatomy of the parts is not within the province of this paper, I shall presume to summarize briefly from the literature so that we may draw certain clinical deductions therefrom. Five vessels contribute to the supply of this region, entering into a free anastomosis about the tonsil, and it is probable that the blood supply is not typical in every case, coming as it does from such varied sources. According to Davis¹ the main arterial supply comes from an anastomosis outside the fossa itself contributed to principally by the ascending palatine branch of the facial and the descending palatine branch of the internal maxillary. The resulting vessel enters the fossa at its upper extremity and, coursing down between the capsule of the tonsil and the aponeurosis of the constrictor, pierces the capsule to enter the substance of the tonsil at about the middle of the fossa, slightly anterior to its center. It is true that this vessel can frequently be seen in the location described after removal of the tonsil and is often responsible when bleeding occurs. The accompanying vein follows the same course.

The ascending pharyngeal artery supplies the mucous membrane and muscles of the upper part of the pillars of the fauces and the soft palate, by its pharyngeal branches, which also supply the superior constrictor muscle.

The tonsillar branches of the facial and the dorsalis linguae branch of the lingual supply the plica and mucous membrane and muscles of the pillars in their lower half or two-thirds and anastomose freely in this region. According to Fetterolf,

branches of these vessels are important in the supply of the tonsil and penetrate the capsule at varying points below the middle. It is certain that bleeding is often encountered low down near the base of the tongue after removal of the tonsil and probably comes from some of these branches which have either been cut in the dissection or severed at their entrance into the tonsil on its removal. Venous drainage in the lower part of the fossa is by means of a plexus of veins, the tonsillar plexus, situated external to the capsule of the tonsil between the aponeurosis of the superior constrictor and the muscle itself, and emptying into the pharyngeal plexus. Anomalies occur here as elsewhere, and it is not unusual to find that an abnormally large artery or vein has been severed and to encounter severe bleeding from it.

It is also well to keep in mind the fact, as mentioned by Makuen,² that the portion of the intrapharyngeal aponeurosis covering the superior constrictor forms at this point the bed of the tonsil and separates it from the muscle. There is a distinct line of cleavage, normally, between the tonsillar capsule, so called, and the aponeurosis and after tonsillectomy the intact aponeurosis should be seen lining the fossa. This ideal may not always be possible of attainment, for in many adult throats which have been subject to frequent inflammations or peritonsillar abscess these fibrous layers are so bound together and to tonsil and muscle that the removal of the tonsil without wounding the aponeurosis is practically impossible. This injury will result in damage to the pharyngeal branches of the ascending pharyngeal artery or to the tonsillar plexus of veins, and being lost in the torn fibers of the muscle they are very difficult to catch in the hemostat. It is therefore highly desirable to keep in mind these relations and avoid, if possible, any damage to the aponeurosis.

With these facts in mind, it appeals to me as being highly desirable to remove the tonsil with the minimum of dissection and by a method that involves the least possible damage to the structures immediately surrounding. This is best done, according to my belief, by the employment of the cold wire cutting from behind forward, slowly, and without preliminary dissection. It is my belief, however, that some cases must be done by dissection in order to accomplish our object.

METHODS EMPLOYED TO STOP HEMORRHAGE

Following the removal of the tonsils, the field should be carefully inspected to ascertain the source of bleeding if any be present. If the bleeding is slight and limited to venous oozing

pressure will control it. If not, or if a pulsating bleeder is seen, its location is carefully ascertained and it is grasped in the tonsil hemostat. Crushing will in nearly every instance prove sufficient, but if there remains any doubt as to its security the bleeding point may be tied. It is my practice to use a suture ligature, as it greatly simplifies the tying and is more secure. Placing a suture at the exact point desired is often very difficult, and I have sought to simplify the procedure by designing a needle holder for the purpose (Fig. 1). The difficulty has been to engage the point of the needle at the proper spot and still have enough curve to carry it through the tissues. By cutting a slot lengthwise through the jaw of the needle holder it is possible to grasp a curved needle at any point in its length, letting the eye extend back through the slot. This also permits grasping the needle with point straight out, usually the best way for this work. One suture will usually suffice, Number 1 catgut being used, but two may be put in if desired or a single mattress suture may be employed. These sutures do not require removal.

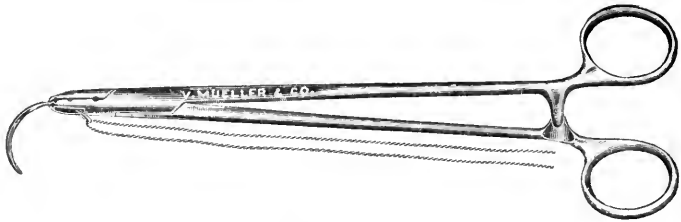


Fig. 1.—Author's needle holder.

SECONDARY HEMORRHAGE

Early secondary hemorrhage, coming on within a few hours after operation, is sometimes the most profuse and may require reanesthetization in a patient not amenable to control. The same principles are applied in these cases as well; however, a sponge in the fossa is utilized for immediate control of very profuse hemorrhage until such time as proper instruments and assistance may be had. Never leave a clot in a bleeding fossa. Great care should be used in watching these patients after operation, especially until they are entirely out of the anesthetic, as it has frequently happened that blood has been quietly swallowed until the patient was nearly exsanguinated before the sudden vomiting of a quantity of fresh blood or the discovery of a bad pulse has called attention to the fact that something was wrong.

They should be placed in the prone position with the head turned to the side and lower than the body so that blood and secretions will flow out of the nose and mouth, and the condition should be carefully watched. Anything less than the best of hospital care is out of place in the management of these cases.

True secondary hemorrhage, coming on several days after operation, is practically never profuse enough to be serious and is usually the result of separation of slough from the exposed surface or comes from the granulations. If it requires control, pressure will usually be found sufficient.

Adenoid bleeding is rarely serious. Packing of the nasopharynx will usually control it, but the bleeding vessel may be located and tied as reported by Adams.³ Beck's method of exposure of the nasopharynx by retraction of the palate with a soft catheter passed through the nostrils and out the mouth is admirable for this purpose.⁴

ACCIDENTS OF ANESTHESIA

This subject has been so well considered in other connections that I shall pass over it very briefly. There is no one thing in the tonsil and adenoid work that is more important than the anesthetic. The anesthetist should be the judge of the anesthetic of choice in each individual case, but my preference is for warmed ether vapor, the patient being sufficiently relaxed to do away with the pharyngeal reflex, but not to the point of abolition of the laryngeal. This point is reached before commencement of operation and is maintained throughout by the use of the warmed ether vapor apparatus. It is surprising how frequently the operation is unnecessarily prolonged by unskillful administration of the anesthetic, a task that should be delegated to none but a skilled and experienced anesthetist.

When operating under local anesthesia practically the only thing to be watched for is the toxic effect of cocaine. As an idiosyncrasy against it does undoubtedly exist in rare cases its use should be very sparing until tolerance has been demonstrated. Novocain and apothasin have been very satisfactory in my hands for infiltration anesthesia, and have given no bad results. Sloughing and edema have been reported following the use of quinin and urea, and I believe constitute a contraindication to their use.

So-called "status lymphaticus" should probably be included under this head. There is no doubt that deaths from this much

blamed condition have occurred, and they are probably in all cases due to arrested respiration from pressure of an enlarged thymus on the trachea, according to Jackson's investigations.⁵ In all cases when respiration ceases during anesthesia and cannot be immediately started the trachea should be opened and tracheotomy tube inserted to reach, if possible, below the probable level of the thymus.

SUFFOCATION

In operating under general anesthesia this difficulty has arisen in numerous cases. Inspiration of pieces of tissue or of quantities of blood may be the cause, an enlarged thymus has been mentioned, and tracheotomy has been necessitated after the use of hydrogen peroxid in the throat to control hemorrhage. Prompt tracheotomy is the only treatment when these emergencies arise, but most of them can be prevented. The operation should be done in the horizontal position when a general anesthetic is employed, the head should be lowered from 4 to 6 inches below the level of the table, careful hemostasis should be observed and with the use of a good suction apparatus to keep the throat clear of blood and mucus most such accidents should be preventable. I am opposed to the use of all chemical hemostatics, especially hydrogen peroxid.

ACIDOSIS

A postoperative complication much considered recently and often met with in these cases is the condition of acidosis or acidemia. It occurs probably more often in tonsil and adenoid cases than in any other one class of patients, but is not as a rule serious. It has been my misfortune to see one fatal case, however, and others have been reported, so that it is not a matter to be considered lightly. Preoperative and postoperative examination of the urine for acetone and diacetic acid with the administration of sodium bicarbonate and glucose per rectum or intravenously both as prophylactic and treatment are the measures of safety.

POSTOPERATIVE PAIN AND SORENESS

One of the most constant and unpleasant manifestations with which we have to deal following the tonsil operation, particularly in adults, is postoperative pain. Coming on almost immediately in local, and as soon as the patient is conscious in general, anesthesia, it persists very severely for varying lengths of time. After trying many remedies I have finally come to follow one routine in all these cases, and I am willing to state positively that

it has been entirely successful in relieving postoperative pain. It consists in the application to the fossa of a solution of 15 grains each of cocain, menthol and phenol in 1 dram of alcohol. It is sometimes, in very bad cases, necessary to repeat the application in an hour or more, but one or two applications are usually sufficient to control entirely the pain that ordinarily follows these operations. In some cases the application seems also to lessen the soreness persisting for some days thereafter, but it is my opinion that it is but slightly influenced thereby. General anodyne treatment is necessary for that purpose, if anything is attempted. It is much easier and more practical to make this application following local than after general anesthesia, but it also seems that it is more necessary, probably because of the entirely normal mental condition of the patient.

SEPSIS

Although slight superficial infection of the exposed tissues is no doubt present in all cases following tonsillectomy it is but seldom that a serious condition supervenes. It has been my experience to have four cases of local infection of some moment. In three, the condition developed into a condition of typical peritonsillar abscess. In the fourth, a suppurative adenitis involving the anterior cervical chain and the submaxillary glands developed, necessitating operation for drainage externally, but with eventual cure. Vanderhoof reports a very similar case.⁶ All four of these patients were operated on under local anesthesia and it has occurred to me that infection might have been carried into the deeper tissues by the needle, particularly since these operations are carried on in a septic field. I have since discontinued the use of deep injections for local anesthesia.

Several deaths from general septicemia have been reported as following the tonsil operation. Dean reports three cases,⁷ and I have personal knowledge of one case with fatal termination occurring in the practice of a personal friend.

Prevention of these infections is a problem for serious consideration, strict asepsis being desirable, of course, but difficult of attainment for obvious reasons. It is our duty, however, to see that septic foci in mouth, nose and sinuses are properly disposed of before operation. The efficacy of antiseptic applications to the fossa is questionable, although many surgeons advocate such measures and routinely apply tincture of iodine, thinking that it does lessen the incidence of infection.

ARTICULAR RHEUMATISM AND ENDOCARDITIS

Acute articular rheumatism has been reported by both Dabney⁸ and Dicky⁹ as following tonsillectomy. I have had one case of endocarditis develop almost immediately following operation under local anesthesia. It is easy to understand how liberation of toxic materials at the time of operation might give rise to these acute infections and careful attention should be given the cardiac condition of our patients both before and after operation, particularly when not doing well during convalescence.

PULMONARY ABSCESS

Manges has reported nine cases of abscess of the lung following tonsillectomy,¹⁰ and others have been reported. It has been thought that this complication is due to inspiration of infected blood, mucus, bits of tissue or pus. If so, careful anesthesia, careful hemostasis, use of the suction apparatus and lowering of the head of the table should eliminate a large majority of them. Richardson, however, has suggested¹¹ that the infection may reach the lung by other channels, notably the lymphatics, or possibly by way of the veins. Such a suggestion seems highly probable, to my mind, particularly as regards the venous channels when we consider the anatomy of the region. The lymphatics, however, can with less reason be included in this theory on account of the fact that all lymph from this region passes through numerous lymphatic glands before it reaches the large vessels, thus being filtered of any infectious material before reaching the lungs. Care to prevent opening the tonsillar plexus should, then, further guard against this form of complication.

Pulmonary infarct, as reported by Scruton,¹² can be explained in like manner, and its prevention would embody the same principles.

PNEUMONIA

This disease has been frequently reported as a complication. It has been my experience to have one case of pneumonia following operation under local anesthesia. Others have had similar experiences in other lines of work, due, probably, to lowered resistance from shock of operation. Under general anesthesia its prevention would embody the same principles outlined for prevention of abscess of the lung. Rapid work, minimizing the amount of anesthetic necessary, and the administration of warmed ether vapor should also play their part.

FOREIGN BODIES IN THE LUNG

Several cases of foreign bodies, such as dislodged teeth, having found their way into the bronchi sound a warning against careless use of the gag, particularly in children who are very likely to have loose teeth that may be easily dislodged and lost.

OTITIS MEDIA

Acute purulent otitis media has followed two of my own cases and has been frequently reported by others. Lowered resistance and local infection following the removal of adenoids were undoubtedly the cause, and prevention is difficult. Moore reports one case of mastoiditis following the same cause.¹³

LYMPHOID HYPERPLASIA FOLLOWING TONSILLECTOMY

Experience has demonstrated that the most careful work will not guarantee against the unexpected appearance of lymphoid tissue in the fossa or attached to the pillars following the healing of a tonsillectomy wound. We all know that most so-called "recurrences" are due to faulty surgical technic, but not all of them. There is no doubt that there is extracapsular lymphoid tissue in the tonsillar fossa, in the pillars, in the plica, and between the base of the tongue and the tonsil, in many cases, and such tissue will undergo hypertrophy following the operation and will sometimes embarrass us by its appearance in a fossa which we considered absolutely clear of tonsillar tissue at the time of operation.

I have been able to demonstrate the presence of extracapsular lymphoid tissue histologically, both in the pillars and in the floor or the fossa; tissue which is structurally identical with that of the tonsil itself, but which is entirely extracapsular and ordinarily would not be removed at operation (Figs. 2 and 3).

DEFORMITIES

Damaged pillars, a torn palate or a uvula inadvertently removed may be included under this head. Most injuries to pillars and palate are the result of carelessness or incompetence. It is true, however, that at times the mucous membrane may be stripped from the posterior pillar and may even carry with it the uvula. This accident is due to the fact that the tonsil is adherent to the pillar and when drawn into the snare brings with it the attached membrane, which is stripped up from the pillar and may even tear the uvula away with it. In these cases the uvula will be found attached to the tonsil by a short strip of

mucous membrane, after removal. Care in freeing the posterior pillar and the use of a small fenestration will prevent this accident. Extreme cases of deformity from laceration of surrounding tissues during operation are likely to result in damage to the voice and interference with palatal function. It is remarkable what serious deformities may exist without apparent interference with vocal function, however, although such serious damage would no doubt prove fatal to a fine singing voice. Excessive scar formation and contracture are likely to follow operation on syphilitics.

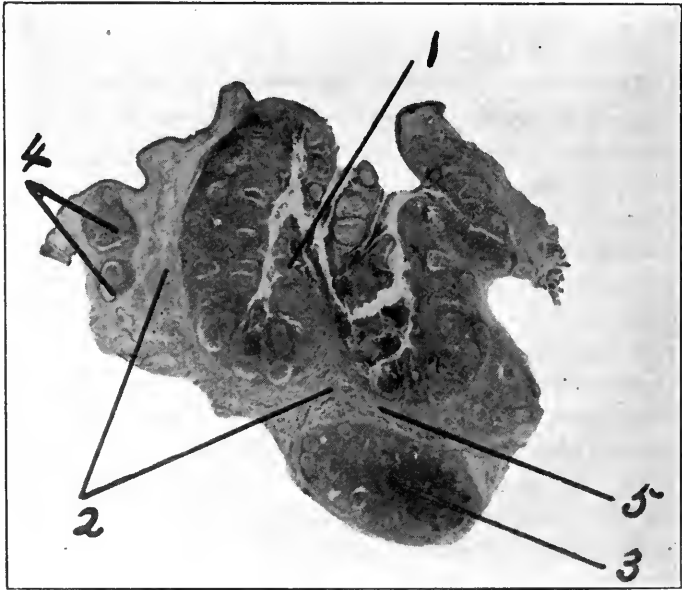


Fig. 2.—Cross-section of tonsil showing the presence of extracapsular lymphoid tissue. (1) Main body of the tonsil, (2) capsule—fibrous tissue, (3) lymph follicle—extracapsular, (4) two small collections of lymphoid tissue just beneath the mucous membrane of the anterior pillar, (5) capsule containing some muscular tissue separating the tonsil and lymph follicle.

Nasopharyngeal scarring is also often seen and results from too violent use of the adenotome, even the eustachian eminence having been sacrificed in certain cases. Tubal troubles are often secondary to these conditions, both palatal and nasopharyngeal, on account of interference with proper action of tubal and pharyngeal muscles.

NEURALGIA AND DRYNESS OF THE PHARYNX

Subsequent to healing of the tonsillectomy wound we sometimes have patients returning with complaint of pain in the

lateral pharyngeal region near the base of the tongue, with dryness. There is no doubt, as has been shown by Matthews,¹⁴ that branches of the terminal portion of the glossopharyngeal nerve may be caught in the scar at the junction of the palatoglossus with the tongue, giving these symptoms. Occasionally a short section of the nerve is exposed at this point during operation, resulting in unusual sensitiveness, with severe lancinating pain, referred along the inside of the lower jaw and lateral pharyngeal wall. This can be quickly relieved by local application of cocain, but will return until such time as the nerve is covered by granulations. I have had one such case in my own practice.



Fig. 3.—Photograph of tonsil showing the presence of numerous lymph follicles in the plica and mucous membrane between the tonsil and base of the tongue.

COMMENT

Many other complications and sequelae have been mentioned in the literature, but they have either been so extremely rare or so entirely unexplainable, or have seemed to me to be so unfairly laid at the door of the tonsil operation that I have not taken the space to mention them.

In conclusion, I wish to state that this paper is not intended to serve as an arraignment of the very valuable and useful procedure under discussion nor as an argument against the performance of these operations, but is presented simply as a sug-

gestion toward the lessening of the number of serious or unpleasant complications to somewhere near the necessary minimum; for we all know that no operation can be undertaken without incurring a certain amount of necessary risk.

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DISCUSSION

DR. T. J. GALLAHER, Denver: The article given by Dr. Baum is very interesting and gives a splendid résumé of the dangers incidental to tonsillectomy. More tonsils are removed by the general practitioner than by specialists, and as suggested by Dr. Large I believe that all facts relating to this operation should be made known. Death from hemorrhage and shocking deformities are still occurring. The proper removal of the tonsil requires skilful and artistic work and certainly should not be made light of. We perform tonsillectomy in children under general anesthesia but in adults local anesthesia alone is used. If the proper points are injected a proper blocking can be secured and the operation should be done painlessly. It is surprising how few men perform tonsillectomy in the adult under local anesthesia, but it is encouraging to note that the number is steadily increasing. For children under general anesthesia the gag should be used in the front of the mouth, as the side gag may dislodge a tooth resulting in its falling into the lung. Of course, keeping the pharynx below the level of the larynx should always be maintained. Very often there is lymphoid tissue below the tonsil which extends up from the pyriform fossa and this should be removed in every instance. If allowed to remain it enlarges and becomes infected growing into the sinus tonsillar. We find that the Sluder instrument will not remove this lower mass in many instances. We remove the tonsil by the method especially adapted to the case. The Sluder method, dissection, with snare, and the cold wire in a fenestra without dissection, constitute our methods.

DR. DAVID STRICKLER, Denver: I think in operations generally on the tonsils the matter of technic is a matter of individual preference. Personally, however, I am using the Beck-Schenck instrument with very good satisfaction to myself in a very large percentage of cases, in some cases having to use the dissection with the snare. I was using the Sluder instrument at the beginning, and when I got my Beck-Schenck instrument for a time I removed one tonsil with one instrument and the other with the other, noting the condition of the two tonsils as they came out, and I concluded, after several operations, that there was no question at all but what I got less tissue outside of the gland by

the snare operation than by the Sluder instrument, and furthermore, there was less soreness, less pain, and less contraction following the Beck operation. I am very much pleased indeed with the Beck operation.

DR. J. J. KYLE, Los Angeles: I am very much pleased to hear Dr. Bane say something about the basement membrane of the tonsil. I think that is the proper term.

Our rule is, and I have experimented a long time, to separate the tonsil from the pillar and take a very small hemostat and place it behind the tonsil and catch the tonsillary artery and vein.

Have you observed an abdominal surgeon take out an ovary and clamp the ovarian artery to prevent hemorrhage? It occurred to me I could do the same thing in a tonsillectomy. If there is any evidence of hemorrhage I put a sponge high up between the pillars and sew them together. I am not going to get up at midnight and go to the hospital to stop a hemorrhage. You moisten a little gauze with petrolatum or iodine and place it high up between the pillars; that will do a great deal better than stitching the pillars together. It is a very simple operation.

Now as to anesthetics, I am opposed to operating on children under local anesthesia. I know you cannot do a good job. It has been shown by experience ever since I began operating, that there is little or no danger in ether when it is given by the drop method—the American method. I always order a general anesthetic for children under the age of 15. You have a better chance to control hemorrhage. You even have a better chance to control hemorrhage in an adult under a general anesthetic, because you have the whole field before you, and all the time you want if you have to use a sponge, or if you have to take up an artery, which is a very delicate thing to do.

DR. JOSEPH C. BECK, Chicago: The paper of Dr. Baum is so thorough that it really leaves very little for discussion, and yet personal experience with the points that he brought out, might be worth while mentioning, also something in regard to things that were not mentioned. For instance, in the anesthetic, of course everybody will do as he thinks best for his method of operating, using a general or local anesthetic in this particular individual. A local anesthetic is certainly best for a good patient, intelligent, and where you have plenty of time to work and you can control it; but where you have an Italian or a Russian, who is wiggling around and will not keep still, you will not choose it; I would not bother with a local anesthetic in a case of that kind. Therefore, I favor anesthesia as introduced by gas, then followed by the tube method and subsequently by the open method.

I wonder if the members of this Society have had the experience that I have had lately, for the past year and a half more than ever, of finding that the ether is very poor. Not only is it poor for anesthetic purposes, but it is poisonous, such collapses as dilatation of the pupil, slow breathing and very startling symptoms coming on, that make you think perhaps Dr. Higginbotham is right. We use the best we can get. Squibb's is the best. I am just bringing that out, that that has been my experiences lately. I have seen the ether was not as good, and we use a great deal more of it.

I was interested in the question of the control of the bleeding with this instrument. I have always used this method, by using the needle in this way, and I find this is a great advantage, that the doctor has brought out here, to put the needle in this way, this particular needle holder. I always ligate the bleeding vessel, by a suture on one side, draw that vessel, using one finger in the mouth and the other one outside. That is a very easy way to do. No one need have any diffi-

culty in tying the vessel, the vessel is tied after the sponge is put in the cavity, and the anterior and posterior pillar brought together and held by the same force for a few moments, and after that the sponge is removed. The patient must leave the table not only dry in the throat, but he must be practically awake. That is the anesthetic I use. The patient is not put to sleep when he is doubly anesthetized.

I had a very interesting case recently, of a child 9 years of age, whose tonsil the assistant could not dislodge with his finger. I said: "Let me try it"; but I could not dislodge it. Then I discovered to my great astonishment not less than a teaspoonful of yellow pus pouring out. This patient was brought in as a regular tonsil and adenoid case. It was very interesting to find that.

Now the question of loose teeth, the anesthetist should be instructed as to false teeth, loose teeth in front, and always before a tonsillectomy, so that the gag, when put in will hold; by the way, the modified gag, which lifts up the lip by a wire, and another one that does not catch the teeth, is the best gag that is made, I think that is Jennings' gag.

Just one more point in regard to the teeth in sepsis, if you have patients who have very bad teeth, send them to a dentist before you do a tonsillectomy.

DR. S. H. LARGE, Cleveland: Speaking of the hemorrhage, I use the same method that Dr. Beck does. Making a cross suture, so it is absolutely impossible to have a hemorrhage following that kind of a ligature.

I heard of a cure the other day that has never been seen in literature, I am sure. We had a physician in one of the suburbs of Cleveland, who attempted to do a tonsillectomy and he got a hemorrhage, so he grasped a great big curved needle about 3 inches long, and he entered the neck outside and attempted to turn it. His idea was to ligate the carotids; the curved needle broke, then he got another and that one broke. By that time they had summoned assistance and arrested the bleeding. I think that was one of the most heroic treatments I ever heard of for hemorrhage.

Speaking of acidosis: I think one of the greatest causes of acidosis is starvation.

DR. OTIS ORENDORFF, Cañon City, Colo.: I have had a few personal experiences that bear directly on the papers of today, that I believe will be of interest.

Regarding coagulation time, and the test for that, at one time I operated on a girl about 10 years old. Her sister was about 14; I operated on both of them. There was nothing of interest in the operation of the elder girl at all. In the second girl, I removed the right tonsil. I always stop the hemorrhage entirely; as usual, it was dry, and then removed the left tonsil and noticed there was some bleeding, and we had a little trouble in stopping it; it was not entirely stopped so when I went ahead with the adenoids and removed those, and by the time we excised the adenoids the bleeding was profuse from them, and she turned out to be a transient hemophilic. During the first part of the operation, the blood coagulated, and by the time we finished the adenoids the blood separated into fibrin and serum; it looked like little black fish worms. We used tampons and everything we could, and I tried Dr. Beck's method of putting the catheter in there for an inspection, but I could not see anything except the blood. The second day she was just about moribund, and still bleeding, when we secured some coagulose and the hemorrhage stopped at once. The little girl recovered, but was in bed for two or three weeks.

Now, I would like to ask Dr. Baum if he uses that new preparation put out by Armour, called cephalin.

With regard to rheumatism, that was brought home to me in the case of my own daughter, about 10 years old, whose tonsils were removed. She had an uninterrupted recovery, but went into a severe attack of articular rheumatism. I promptly put her to bed and used ice, and she made a recovery in two or three weeks, without any complication. But since then, whenever I operate for rheumatism—and by the way, most of our tonsil operations are for rheumatism—I always keep those patients in bed, so that the latent rheumatism will not be stirred up by the operation.

Now we have all gone through these different methods in tonsillectomy, I have tried all of them, as you have without a doubt, and I have worked out a modification that seems to me to be somewhat a happy medium—a successful modification of the different methods—that is, I use a very heavy snare, and use the heaviest wire that will work in that snare, No. 10, and I use the cheek retractor gag with the general anesthetic, which is better than any other I have used. The inspection of the throat is marvelous. If you have never used one, you will be surprised. I take the heavy snare and conform the wire more or less to the particular case; I adjust that with my fingers, and can take out just the amount of tissue that is desired.

DR. HARRY A. SMITH, Delta, Colo.: Let us forget we are specialists and when you have got an operation to do, be a surgeon, and do not make anybody's incision except your own. You are a surgeon, you are going to have to do a surgical operation, and you meet the requirements as they come up, whatever they happen to be.

But I got up to defend quinin just a little bit, because I hear it abused every little while. I presume that everybody will say that quinin causes sloughing, except myself. I have used quinin for seven years. I do two-thirds of my tonsillectomies under local anesthesia, and I have had two cases of sloughing in the same patient. This patient was a bad patient, to begin with. Quinin is slow, and that is the only objection to it. It is absolutely safe, and your afterhemorrhage is less. I take 2 grains and put it in a half ounce of solution and boil it an hour. If you are going to do your tonsil operation in a hurry, do not use quinin. But you take a half hour to anesthetize your patient, and it is absolute, and you have no after-pain. I have taken children 10 years old and 9 years old that did not have any after-pain, it was healed up, no pain whatsoever in young children. In doing local anesthetic work, there is a bit of mesmerism attached to it. If you do not take your time and get your patient right, you are going to have trouble.

If somebody else would tell me what they think about quinin, I would be much obliged.

DR. THOMAS E. CARMODY, Denver: This has developed into such a general discussion; it seems to me that nearly everything has been said, and Dr. Baum has covered the ground, I am sure, very thoroughly.

I notice the mentioning of warm ether; when any body mentions warm ether they mean ether vapor. We do not mean that. The vapor is warm, not simply vaporized. Dr. Roehrig has an operation that warm vapor. After it has been vaporized it comes out of the tubes at a temperature of from 106 to 110 F., and if you have it that way you get better results. I believe that possibly has something to do with our not having trouble with our ether. Also, the suction, I believe, has a great deal to do with our not having pulmonary abscess, despite the fact that pulmonary abscess may come from some other cause.

I had one case recently that had pneumonia following a nasal operation, which was done under a general anesthetic on a physician, who did not want to stand a local anesthetic. That was due to just the point that Dr. Baum brings up, that the patient be put on the side, or put so that the head is turned to the side. I always have them put them on the side and put a pillow on their back, so that they are entirely on their side. Nevertheless, they sometimes lapse into unconsciousness.

There is just one more point I wish to cover, and that is the teeth. Dr. Gallaher covered the point exactly. The side gag is what knocks the teeth out. If you have the front gag, or any modification of it, you will see the teeth are loose, but it is the side gag that loosens the teeth in children around 9, 10 and 11 years of age, in whom the temporary molars are ready to come out, and that side gag, with the patient struggling, if they are not entirely under, will loosen the teeth.

DR. R. P. SCHOLZ, St. Louis: There really is nothing that I have to add to these very interesting papers, so will merely relate some of my experiences.

Ordinarily, I use nitrous oxid-oxygen, except at the St. Louis City Hospital, where I use ether, because at this institution I instruct interns, and find that for these beginners, gas is too short an anesthetic. I sometimes use a local anesthetic. I realize that many are opposed to gas as an anesthetic, but my experience has been so satisfactory that I am continuing its use. I wish to relate one unusual experience I had recently with it. A boy, about 9 years of age, was anesthetized by my assistant, who is recognized as an expert anesthetist. It is the latter's practice to administer nitrous oxid just up to the point of cyanosis, then oxygen is switched on so that for the rest of the anesthetic there is practically no visible cyanosis. In this particular case the young chap became cyanotic as usual, and except for several deep sighs nothing unusual was noted. When the oxygen was switched on, the slight cyanosis did not clear up. No change had been noted in the pulse, however, and as our suspicions for the welfare of the child had not been aroused, I removed one tonsil (Sluder method). About this time we realized the child was not doing well; there was absolutely no bleeding although the pulse still remained good; the respiration had ceased. Immediately artificial respiration was resorted to, oxygen administered, sphincter ani dilated, but only after much time and effort did the patient finally begin to breathe. After he had come around to the stage where he began to struggle, I removed the other tonsil and adenoid. With the removal of these there was no bleeding; the impression was like that of organs having been removed from a cadaver. The unusual feature in this case followed: Instead of the child coming out of the anesthetic he passed into a raving delirium, in which he remained for more than five hours, struggling and screaming incessantly. Only during the last fifteen minutes of the delirium, did the symptoms pass off gradually. From this on the recovery was uneventful. I believe this was a central intoxication by nitrous oxid in a patient with an unusual idiosyncrasy.

In Dr. Baum's paper he spoke rather slightly of serum as a means of controlling hemorrhage. I had one case which convinced me that normal horse serum is certainly a very valuable remedy in hemorrhage. Nov. 28, 1912, Dr. J. C. Falk, referred a man to me who gave a true history of hemophilia. The man had almost bled to death on several occasions, bleeding over periods of four weeks at a time, from tiny abrasions on his hands, slight gum wounds, etc. When brought to me he was suffering from epistaxis. In the beginning I thought certainly I could control the hemorrhage. I used all the local remedies that we employ in such cases, but in spite of these the hemorrhage con-

tinued, the blood oozing through even the tightest tampon. After about a week's work in vain, I recalled that a writer had suggested serum in hemorrhage as a controlling agent. I injected 30 c.c. subcutaneously and it worked like a charm. After that the patient would go for about six months before hemorrhage again would set in, which was controlled in like manner by the serum. So the patient continued for about five years; until his death which was caused by pneumonia.

After the removal of tonsils in patients who have had general infection (rheumatism) I have seen rheumatic attacks flare up, and I believe, as does Dr. Beck, that this is a favorable rather than unfavorable sign. I have often consoled patients in whom I have noted it.

DR. JAMES J. PATTEE, Pueblo, Colo.: With reference to hemorrhage, many valuable points have been brought out, but the question of hemorrhage is dealt with every time one deals with open vessels, and you never can be certain that you will not have trouble. As a routine, I am unmindful of a little early hemorrhage, much as I am of a little early hemorrhage in the mastoid operation, because there is a natural tendency for it to cease. Tonsil hemorrhage has a tendency to cease, if given a little time. Mastoid flap hemorrhage ceases on applying retractors and waiting a little. Experience has shown that unless bleeding is a little unusual, it will cease in three or four minutes. If bleeding continues, the artery may be ligated. I have found it very much easier to place a compress of gauze into the fossa and lace the pillars over it. One or two sutures will hold the compress. The compress is efficient, easy and quickly put in. If the compress is too large, it tends to slip out. I use a slender needle holder and rather a long needle. The long needle is used because if it should break, it is easier to get the fragments than would be the case if a short, fine needle were used. The compress is removed the following morning. I do not use the suction apparatus because I prefer the use of sponges.

DR. JOSEPH E. SAWTELL, Kansas City, Mo.: Dr. Baum has given us a splendid paper but there are some things I have learned to do differently.

With reference to injection for local anesthesia, I formerly used superficial but changed to the deep injection with results far more satisfactory. The tonsil is seized with a sharp or bayonet forceps, freed from any of its coverings and traction is made toward the median line until a sulcus is observed which marks the external boundary of the tonsil. In this depression three injections are made; one above the velar lobe, one posterior to the center and another at the lower portion, in all about 2 drams for each tonsil. As soon as the needle passes well through the anterior pillar there is no further resistance until it reaches the posterior pillar. In this space the nerve supply both to the pillars and tonsil is blocked by the solution and anesthesia is instant. I have not found the after-pain greater following this procedure than after general anesthesia.

Regarding the control of hemorrhage, this was more annoying to me than anything else when I began the practice of laryngology. The methods in use then were crude and cumbersome and in seeking a better method I adopted torsion, which has continued to prove satisfactory. I have now performed more than 10,000 consecutive tonsil enucleations in which I have not in a single instance tied a vessel, stitched the pillars, used a compression instrument of any kind or resorted to any device other than torsion. Every bleeding vessel is thus controlled at the time of operation. In about 1 per cent. of these cases after-hemorrhage occurred. When a thorough curetting of the blood clot from the tonsillar fossa failed to control it, which was successful in at least 50

per cent. of the cases, torsion or crushing the bleeding vessel was resorted to, with the final result that there was not a single fatality in this series of cases.

DR. HAROLD BAILEY, Springfield, Mo.: There are all sorts of tonsils and a great many methods have been devised for their removal. Each of us has had more or less experience with the different methods. It seems to me that I have tried every instrument and method that has ever been devised for tonsillectomy. Some of the best tonsil work I ever did in the days before the snare and Sluder instruments came into general use I did with my fingers, enucleating the tonsil very much as the general surgeon does the prostate. I still use this method occasionally in children. It is especially applicable in those cases where the nurse has packed your grip and left out your tonsillotomes. The removal of the tonsil by means of the actual cautery, as advocated and practiced by the late Dr. Pynchon and others, has something to be said in its favor. It is bloodless, and I find it still applicable in old people with sclerosed vessels in whom one has every reason to expect serious hemorrhage. From the standpoint of the operator it is ideal. The convalescence, however, is both prolonged and painful. At the present time the snare is a very popular instrument and I think deservedly so. I like it especially well in adults. In children I prefer the Sluder operation. For me it is easier and quicker than any other method. The entire tonsil comes out cleaner and the adjacent structures are less likely to be injured. It is a good idea when we first examine our patient and decide on tonsillectomy, to study the tonsils carefully and plan the method and procedure best suited to their removal.

Dr. Higginbotham spoke of doing tonsil work without any anesthetic whatsoever. This, of course, is not a new method. They have been practicing it in Germany for years. I do not think it will ever become popular in this country. The patients do not take to it kindly, nor do their friends and relatives.

One word in regard to hemorrhage: as Dr. Sawtell said, it is usually quite easy to stop tonsil bleeding. There are some cases, however, in which it is not so simple. I had an experience about three months ago in the case of a woman about 60 years of age who had badly sclerosed vessels. I removed a tonsil for the relief of chronic and persistent soreness. I found a small collection of pus immediately outside of the capsule, a chronic peritonsillar abscess. I used the snare in this case and the hemorrhage was most profuse. I applied forceps but the tissues were so diseased they tore before I could put on a ligature. I used deep sutures and sutured the pillars as well which checked but did not stop the bleeding, and I was obliged to ligate the external carotid before I could leave this patient with safety.

DR. HARRY L. BAUM, Denver (closing): Gentlemen, I wish to thank you for the very interesting discussion of my part of this program, and to say that many interesting things have been brought out, some new, and also to add, in self defense, that it was impossible for me, in the length of my paper, to mention the many things which have been mentioned in the discussion. I am glad that they were brought out for that reason, as my paper was entirely too long anyway, although it seemed to me that I could not compress it into less space and still touch on each point that I had in mind.

Dr. Large mentions the influence of fasting on acidosis. I never instruct my patients to fast in preparation for a tonsil operation and I think his point an important one. As to the theory that phenol is neutralized by the action of alcohol, I am not a chemist, but I believe

that such is really not the case, although I know that alcohol is used as an antidote for phenol poisoning and that such is the opinion generally held by the profession. Alcohol has the effect of dissolving the phenol and it is as a solvent that I have used it and the mixture is made in the strength of 15 grains to the dram of alcohol, so of course that makes it a good deal stronger than Dr. Large understood it to be. If we only used 15 grains to the ounce of alcohol it would not be sufficient to have any effect.

As to Dr. Smith's experience in the use of quinin, I am much interested. I have never used quinin and I freely admit that what I said in my paper about quinin was said on the strength of the experience of other men. It has seemed to have caused trouble, consequently I mentioned it in that way. But if Dr. Smith has had good results, that is a word in its favor.

Dr. Carmody mentions the definition of warm ether. I mean exactly what Dr. Carmody does and I agree with him absolutely. Our anesthetics are given by the same man with the same ether machine and it is the vapor that is warmed, not the ether before vaporizing. As to coagulation time, it certainly has its place, but my main point in regard to it is that most of our hemorrhages have nothing to do with coagulation time. They are due to the mechanical fact that an artery has been severed and it makes little difference whether the patient's blood coagulates in three minutes or a few seconds, it is the same. It has its place, however, and is of value. Dr. Carmody has had a great deal of experience with it, in fact has written an excellent paper on the subject.

Dr. Scholz says he thinks I spoke too lightly of the use of serum. I did not mean to. I think, in the absence of possibility of transfusion, that the use of normal serum is the measure of choice in dealing with cases not amenable to control by mechanical means, such as the hemophilias and blood dyscrasias.

Dr. Sawtell mentions deep injection. I disagree with him in his definition, because I do not believe that when you pull the tonsil out until you have a very thin area of attachment before making your injection that you are giving a very deep injection, although it can be so spoken of.

I wish to thank Dr. Beck for his complimentary remarks regarding the needle holder. It is so simple a modification that I hesitated to present it, but it has been of great assistance to me in tying these bleeding points and has made the procedure an easy one and I have never seen it mentioned in any other connection. It is true that torsion will control almost any arterial bleeding but I feel safer when I have seen them securely tied and, as Dr. Kyle says, that I am not going to be called out of bed to stop a postoperative hemorrhage.

DR. WILLIAM C. BANE (closing discussion): Dr. Smith spoke of quinin. I have used it some with satisfactory results, but I have drifted away from the use of it. I have never had any bad results from it. However, possibly I did not wait long enough for the full anesthetic effect, before I proceeded to operate.

As to apothesine, Dr. Smith has spoken of after-hemorrhage. I have used apothesine with adrenalin five times locally, and in three of the cases I had after-hemorrhage. I remember one case that when I started to operate I had an oozing of red blood and a good deal of edema. Edema interferes very much with the operation.

OSTEOMA OF FRONTAL SINUS

R. A. REEVE, M.D.

TORONTO, CAN.

If Friedenbergl, in making public a similar case a number of years ago, felt moved to offer an apology in view of the number of cases already on record, what excuse can the speaker offer on the present occasion!

Well, not to waste words or time one can at least offer the reason that it is desirable to make statistics as correct as possible, for, as we all know, they have been regarded as proverbially inaccurate—not, perhaps, so much for what they contained as for their omissions. For example, the long list of osteomas of the frontal sinus and orbit collected with painstaking care and published by Lagrange in his large work would have been more complete if it had included the osteoma of the frontal sinus and orbit removed by myself in 1881 and not reported, and the present one now published.

REPORT OF CASE

History.—Miss M., aged 23, consulted me on April 8, 1895. Her mother had noticed a slight swelling at the upper, inner angle of the right orbit one year previously, but some friends had spoken of a fulness at the lower part of the forehead before this. It was rather the instinctive dread of what might happen if the matter were not attended to than the experience of difficulty or pain that led the patient to seek advice. Her sight was excellent and there had been no diplopia.

Status Presens.—There is a distinct prominence at the lower part of the forehead on the right of the vertical mesial line and extending outward along the brow to about its middle, and downward nearly to the inner canthus, with moderate edema of the upper lid. V—R. E., 20/20; V—L. E., 20/15. With +0.75 sph., 20/15. Field of both eyes was normal; slight perineural hyperemia of right optic disk; no diplopia. Excursions of right eye upward and inward were somewhat curtailed. Eyeball displaced one-eighth inch down, one-eighth inch out, and forward one-eighth inch. Palpation yielded a distinct bony feel from a slight sulcus at the orbital rim down to the inner canthus outward to the junction of the inner and middle thirds of the brow, and well backward to the inner side of the eyeball.

Diagnosis.—In view of the history, and with some experience gained in a previous case, I did not hesitate to give a diagnosis of bony growth in the frontal sinus, and to advise removal. The operation was agreed to, and was done without any delay except for the ordinary preliminary preparation.

Operation.—An incision was made to the bone just below the orbital rim from the junction of the middle and outer thirds of the brow and curved downward to the inner side of the lacrimal sac. Special care was taken to avoid the levator of the upper lid. The soft parts were well separated and the anterior wall of the frontal sinus was fully exposed. Careful explora-



Figure 1.

tion was made through the depressed bony floor of the sinus (roof of orbit), when the tumor was found so firmly impacted that removal of the anterior wall of the sinus was decided on. This was readily effected by chiselling it away for an area of 20 mm. upward from the orbital rim and 20 mm. laterally. The growth was thus well exposed and by careful use of the chisel at the sides and some prying it was brought away. A small collection of mucous secretion was removed from the lower part of the cavity and all debris from the upper part; also the lining was made as clean and aseptic as possible. The wound was closed by sutures, some of which were deeply placed so as to unite the fascia and periosteum of the roof of the orbit and of

the frontal region, a rubber drainage tube being inserted at one angle. The healing was uneventful. The stitches and the rubber were removed at the end of a week.

Results.—After a few weeks of varying muscle imbalance without diplopia, except when looking downward and to the right (at 45 degrees), the eyes settled down to a hyperphoria of 1 degree, with V — R. E. 20/15.

REMARKS REGARDING THE DIAGNOSIS

If I had had the benefit of the roentgenogram of the parts I might have done the Golovine osteoplastic resection. However, the growth pressed so tightly against the anterior wall of the sinus that the roentgen ray might not have helped me out. Per-

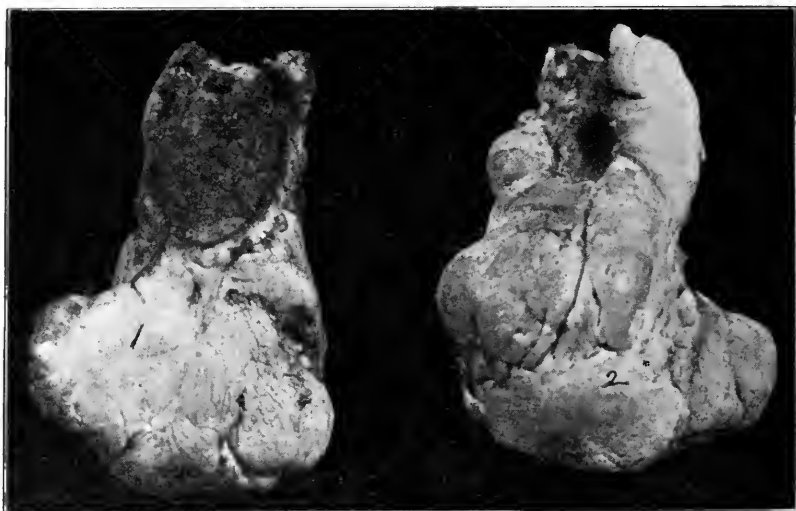


Fig. 2.—Ivory exostosis, $1\frac{1}{2}$ in. vertically, 1 in. ant. posteriorly, $\frac{3}{4}$ in. laterally. 1 nasal side; 2 antero-temporal view.

haps one may be pardoned for not securing a roentgenogram, as the procedure for that purpose was but little in vogue at that date. It should always be resorted to as an aid to diagnosis: It enables one the better to decide where the tumor is free from its surrounding or adjoining walls and to locate the base; a point of importance in the attempted removal of the tumor. In order to learn the size and relations of the latter both front and lateral views should be taken as in Veasey's case.¹

1. Trans. Am. Ophth. Soc., 1916. J. Tweedy, Trans. Ophth. Soc. N. K., 1905, is reported to have said he would not have operated on his case (1881), which ended fatally a month after operation, if the roentgen ray had been available.

ETIOLOGY

The idea of trauma as the dominating causative factor, directly or indirectly, has not been sustained by an analysis of the larger number of cases gathered in recent years. Probably one is not far astray in saying that these osteomas are the result of the unhappy accident of anomalous development more often than of trauma *per se*.

The rough areas on the sides and at the top of this tumor, marking the sites of attachment, confirm Virchow's view, that such osteomata originate from the diploë of the frontal bone.

OPERATIVE PROCEDURE

The least trauma compatible with successful removal should be the rule. To this end the method successfully adopted by Maisonneuve, 1861, Ann. l'Oculist, p. 51, and advocated and practiced by H. Knapp, 1880, Archives of Ophthalmology, should be followed: After denuding the growth of its periosteum, which is saved as far as possible, one chisels around the base of the osteoma, "not attacking it from the surface or chiselling or sawing it off piecemeal" (H. Knapp). In the writer's case (1881) of successful removal of an osteoma of the frontal sinus and orbit, a strong curved *director* proved very useful in severing attachments and avoiding injury to the dura mater. The value of the *drill* when skilfully used, in effecting safe removal of part of a bony growth where the base has to be left, is well shown by Edward Jackson's case, 1892, Trans. Am. Med. Assn.

PROGNOSIS

The "let-alone" or "dilly-dally" policy has been proved by experience not to have been the best. For example, Birch-Hirschfeld's mortality was as follows: Frontal, unoperated, 48 per cent., operated, 13.6 per cent.; ethmoid, unoperated, 80 per cent., operated, 12.7 per cent.; sphenoid, unoperated, 100 per cent., operated 33 per cent. The inference is plain.

DISCUSSION

DR. EDWARD JACKSON, Denver: About the only profitable discussion of this subject, would be to discuss our own experiences. In a rare condition like this, every case is of value. I have had but one case, and that I reported a good many years ago, and I have seen two others in consultation with other men. The specimen I removed is here presented. It was an incomplete removal. The upper edge of the growth was drilled through and broken off. There was a mass of firm, ivory exostosis left. But five years after there had been no regrowth whatever.

Dr. Reeve referred to the diplopia following the operation. In my case the eyeball was displaced 10 mm., and following the operation took its normal position and the diplopia disappeared in a few days. The growth originated apparently from the junction of the frontal sinus and the ethmoid. The chief attachment was at the base of the frontal sinus. The displacement was forward, down and slightly out. There was diplopia following the operation, which ceased in ten days, and in a month the balance was practically normal.

I can heartily agree with Dr. Reeve about making the traumatism as slight as possible. My case seemed to indicate that a small portion of the growth could be left. This may make the operation safer, as there is not likely to be a regrowth. The patient was 19, and the growth started five years before.

DR. R. A. REEVE, Toronto: In regard to the regrowth of osteomas where the base or a portion has been left, I may cite a case which was under my care a number of years ago—ivory exostosis of the external auditory meatus, growing from the mastoid wall forward until it impinged against the anterior wall causing deafness and other annoyance, and also pain during mastication. The tumor was removed by first perforating it at the center with a small and very hard drill-point attached to the flexible arm of an electric dental engine; and then enlarging the opening with burrs until the ivorylike base was flush with the skin of the meatus. When the patient was seen after a long interval there was no sign of return.

A CASE OF HARE LIP AND DOUBLE CLEFT PALATE

T. E. CARMODY, M.D.

EXHIBITION OF PATIENT

One of the features of the operation was invented by Beck, although I have performed it myself three times previous to this operation, to bring down the inferior turbinates to close a portion of the cleft. The patient came to me at seven years, and he is now nine. I have not been able to close the palate entirely, but nearly so (illustrates).

DISCUSSION

DR. BECK: Dr. Carmody was kind enough to credit me with this operation of transferring the inferior turbinate into the cleft palate. This shows where the rhinologist enters the field of plastic surgery of the palate. One of the most difficult things to do is to close an old perforation in a hard or soft palate. It has been attempted many times in an amateur way, and no matter what procedure is used it is a failure. The blood supply is the essential thing, and in scar tissue the blood supply is practically negative. As shown here, transfer of the inferior turbinate will bring this so the edges may be brought together. I have a week ago operated by a new method recommended by Federspiel of Milwaukee, using plates and wire to draw together. This is a good way in old perforations, and Dr. Carmody will probably use this finally to close this perforation. The part of the inferior turbinate is left attached and brought down and I congratulate him on the success of this case. My cases were all old, 25 or 30 years.

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▽ Indicates serving in the U. S. A. Medical Corps. Corrected to Oct. 19, 1918. Members in service not so designated, also those whose address is incorrect or incomplete, or those whose specialty is not given are requested to notify the Editor, so that correction can be made in the next volume. O (Op), indicates ophthalmologist; A, indicates aurist; L, indicates laryngologist; R, indicates rhinologist. Combinations indicate two or more specialties.

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| Barker, Olin G. A. | Johnstown Trust Bldg., Johnstown, Pa. | Op. |
| Baron, Frederick Stewart | 511-514 Peoples Bank Bldg., Zanesville, O. | OALR |
| Barnes, John Henry | Enid, Okla. | OALR |
| Barnes, L. E. | 711 West 69th St., Chicago, Ill. | OALR |
| Barnhill, John F. | The Pennway, Indianapolis, Ind. | ALR |
| Bartholomew, A. C. | Van Wert, O. | OALR ▼ |
| Battle, K. P., Jr. | Raleigh, N. C. | OALR |
| Baum, Harry L. | 264 Metropolitan Bldg., Denver, Colo. | ALR |
| Beach, Sylvester J. | 283 Water St., Augusta, Me. | OALR |
| Beane, Geo. W. | Jenkins Arcade Bldg., Pittsburgh, Pa. | OALR |
| Beattie, Robert | 608 Gas Office Bldg., Detroit, Mich. | OALR ▼ |
| Beatty, Hugh Gibson | 150 East Broad St., Columbus, O. | OALR |
| Beaudoux, H. A. | 140 Lowry Bldg., St. Paul, Minn. | Op.A |
| Beck, Jos. C. | 2551 N. Clark St., Chicago, Ill. | ALR ▼ |
| Becker, Harvey M. | 49 S. 4th St., Sunbury, Pa. | OALR |
| Bedell, Arthur J. | 354 State St., Albany, N. Y. | Op.A |
| Beebe, C. S. | 173 Wisconsin St., Milwaukee, Wis. | OALR |
| Beem, Ione F. | 235 W. Washington St., Chicago, Ill. | OALR |
| Beggs, Wm. F. | 17 Fulton St., Newark, N. J. | OALR |
| Behrman, Michael | 727 Madison Ave., Covington, Ky. | OALR |
| Bell, Mace Hudson | Vicksburg, Miss. | OALR |
| Bellin, Joseph | Green Bay, Wis. | OALR |
| Bennett, A. G. | 26 Allen St., Buffalo, N. Y. | Op. |
| Bennetto, Frederick R. | 172 Woolwich St., Guelph, Ont. | OALR |
| Bergeron, J. Z. | 104 S. Michigan Ave., Chicago, Ill. | ALR |
| Bernatz, Clarence F. | 1105 Park Bldg., Pittsburgh, Pa. | OALR |
| Bernstein, Ed. J. | 1212 Kresge Bldg., Detroit, Mich. | OALR |
| Berry, David F. | Hume-Mansur Bldg., Indianapolis, Ind. | ALR |
| Bettison, David L. | 306 Southwestern Life Bldg., Dallas, Tex. | OALR |
| Bigelow, Frederick Norton | 265 Benefit St., Providence, R. I. | ALR ▼ |
| Binkley, Roy S. | 501-2 Schwind Bldg., Dayton, O. | OALR |
| Birkett, H. S. | 252 Mountain St., Montreal, Canada | OALR |
| Bissell, Elmer J. | 75 S. Fitzhugh St., Rochester, N. Y. | Op.A |
| Blaauw, E. E. | 327 Franklin St., Buffalo, N. Y. | OALR |
| Black, J. H. | Lebanon, Ind. | OALR |
| Black, Melville | Metropolitan Bldg., Denver, Colo. | Op. |
| Black, Nelson M. | Wells Bldg., Milwaukee, Wis. | Op. ▼ |
| Black, W. D. | Metropolitan Bldg., St. Louis, Mo. | OALR |
| Blackham, Geo. E. | 338 Central Ave., Dunkirk, N. Y. | OALR |
| Blair, Wm. W. | Diamond Bank Bldg., Pittsburgh, Pa. | OALR |
| Blake, C. J. | 226 Marlborough St., Boston, Mass. | A |
| Blake, Wm. Ford | 240 Stockton St., San Francisco, Calif. | Op. |
| Blakesley, T. S. | 638 Lothrop Bldg., Kansas City, Mo. | OALR ▼ |
| Bledsoe, R. W. | 1005 Madison Ave., Covington, Ky. | OALR |

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|-------------------------|---|--------|
| Bliss, Chester B. | 411 Columbus Ave., Sandusky, O., | OALR |
| Blue, Julian B. | 1224 Exchange Bldg., Memphis, Tenn., | OALR |
| Blum, Henry Nathan | 830 Canal St., New Orleans, La., | Op. |
| Bobb, Earl V. | Mitchell, S. D. | |
| Boeckmann, Egil | Lowry Bldg., St. Paul, Minn., | OALR |
| Boehringer, H. Winfield | 1811 S. 22d St., Philadelphia, Pa., | Op. |
| Boiler, Wm. Fred | Iowa City, Ia., | OALR |
| Bone, Herman David | 29 Parker St., Gardner, Mass., | OALR |
| Boner, Geo. W. | Ft. McHenry, Md. | |
| Bonner, Horace | Reibold Bldg., Dayton, O., | OALR |
| Bonser, Wm. O. | 314 Colton Bldg., Toledo, O., | OALR |
| Boot, Geo. W. | Century Bldg., Evanston, Ill., | OALR |
| Bordley, James, Jr. | 330 N. Charles St., Baltimore, Md., | Op. ▼ |
| Bowles, F. J. | 121 W. 93d St., New York, N. Y., | OALR |
| Boyce, Geo. H. | Iron Mountain, Mich., | OALR |
| Boyce, Wm. | Escanaba, Mich., | OALR |
| Boyd, E. T. | 909, 17th St., Denver, Colo., | OALR |
| Boyd, Geoffrey | 167 Bloor St., E., Toronto, Canada, | ALR |
| Bradfield, J. A. L. | Main and Fifth Sts., La Crosse, Wis., | OALR |
| Brainerd, John B. | 419 Boylston St., Boston, Mass., | ALR |
| Brawley, F. E. | 7 W. Madison St., Chicago, Ill., | OALR ▼ |
| Bray, Elwyn R. | Lowry Bldg., St. Paul, Minn., | OALR |
| Breaks, L. Z. | Terre Haute Trust Bldg., Terre Haute, Ind., | Op. |
| Briggs, Wm. E. | 1005 K St., Sacramento, Calif., | OALR |
| Britten, G. S. | 903 University Blk., Syracuse, N. Y., | OALR ▼ |
| Brobst, Chas. H. | Observatory Bldg., Peoria, Ill., | OALR |
| Brodie, George M. | Woodstock, Ont., | OALR |
| Brodrick, Frank Wilson | Sterling, Ill., | OALR ▼ |
| Brooks, Everett H. | Appleton, Wis., | OALR |
| Brose, L. D. | 501 Upper First Ave., Evansville, Ind., | OALR |
| Brown, Clayton M. | 510 Delaware Ave., Buffalo, N. Y., | ALR |
| Brown, E. J. | 39 Syndicate Blk., Minneapolis, Minn., | OALR |
| Brown, Edmund T. | 30 Church St., Burlington, Vt., | |
| Brown, Henry Alexander | 610-618 Medical Bldg., Hyde and Brush Sts., San Francisco, Calif., | OALR |
| Brown, H. H. | 31 N. State St., Chicago, Ill., | OALR |
| Brown, J. E. | 239 E. Town St., Columbus, O., | OALR |
| Brown, J. Mackenzie | 1002 Brockman Bldg., Los Angeles, Calif., | ALR |
| Brown, Louis Emmitt | 406 Everett Bldg., Akron, O., | OALR |
| Brown, Richard H. | 7 W. Madison St., Chicago, Ill., | OALR |
| Brown, S. H. | 1901 Mt. Vernon St., Philadelphia, Pa., | Op. |
| Brown, W. Harold | Edmonton, Alta., Manitoba, Can., | OALR |
| Brown, Walter B. | 632 Union Arcade Bldg., Pittsburgh, Pa., | |
| Broyles, Charles J. | Johnson City, Tenn., | OALR |
| Bruder, Joseph | 78 W. 114th St., New York, N. Y., | OALR |
| Bruère, Gustave E. | Portland, Ore. | |
| Bruner, Wm. E. | The Guardian Bldg., Cleveland, O., | Op. |
| Brust, Herbert O. | 720 S. Crouse Ave., Syracuse, N. Y., | ALR |
| Bryan, W. M. C. | 303-307 Metropolitan Bldg., St. Louis, Mo., | ALR |
| Bryant, A. G. | 502 Beacon St., Boston, Mass., | |
| Bryant, W. S. | 19 W. 54th St., New York, N. Y., | ALR ▼ |
| Buck, Robert H. | 15 E. Washington St., Chicago, Ill., | ▼ |
| Buckley, J. H. | Fort Smith, Ark., | OALR |
| Buckman, E. V. | 72 S. Franklin St., Wilkes-Barre, Pa., | OALR |
| Buckner, R. G. | Asheville, N. C., | OALR |
| Bull, Edward Leonard | 2 Madison Ave., Jersey City, N. J., | OALR |
| Bullard, Wm. L. | Columbus, Ga., | OALR |
| Bulson, Albert E. | 406 W. Berry St., Fort Wayne, Ind., | OALR |
| Burch, F. E. | 935 Lowry Bldg., St. Paul, Minn., | A ▼ |
| Burke, Thos. A. | 536 Rose Bldg., Cleveland, O., | OALR |
| Burleson, John Hill | San Antonio, Tex., | OALR |
| Burns, Horace R. | 317-19 Mack Bldg., Denver, Colo., | OALR |
| Burns, Stanley Sherman | 201 Humboldt Bldg., St. Louis, Mo., | ALR ▼ |
| Burroughs, George M. | 3 Broad St., Danielson, Conn., | Op. |

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| Burton, Frank A. | 404 Watts Bldg., San Diego, Calif., | ALR |
| Butt, M. M. | 1701 Chestnut St., Philadelphia, Pa., | Op. |
| Byrnes, Harry F. | 4 Chestnut St., Springfield, Mass., | OALR✓ |
| Byrnes, M. Robert. | Crete, Neb., | OALR |
| Cain, Charles M. | 703 Hume Mansur Bldg., Indianapolis, Ind., | OALR |
| Caldwell, Robert. | State Natl. Bank Bldg., Little Rock, Ark., | OALR |
| Calhoun, F. P. | 833 Candler Bldg., Atlanta, Ga., | OALR |
| Callan, Peter A. | 452 Fifth Ave., New York, N. Y., | Op. |
| Campbell, C. H. | Columbus, Neb. | ✓ |
| Campbell, Don M. | Peter Smith Bldg., Detroit, Mich., | OALR |
| Caplan, Leo. | 4500 Olive St., St. Louis, Mo., | ALR |
| Capron, Franklin P. | 118 Angell St., Providence, R. I., | OALR |
| Carleton Elmer Howard. | Hanover, N. H., | ALR |
| Carleton, Ralph. | 10 Temple St., Springfield, Mass., | Op. |
| Carmody, T. E. | 520 Metropolitan Bldg., Denver, Colo., | ALR ✓ |
| Carney, A. C. | 111 N. Third St., Hamilton, O. | |
| Carpenter, E. R. | Stanton and Wyoming Sts., El Paso, Tex., | OALR✓ |
| Carr, Geo. W. | 30 S. Franklin St., Wilkes-Barre, Pa., | OALR |
| Carroll, Frank. | Ely Block, Cedar Rapids, Ia., | OALR |
| Carroll, Geo. G. | 614 Main St., Rochester, N. Y., | OALR |
| Carson, W. E. | 5118 Jenkins Arcade Bldg., Pittsburgh, Pa. | ✓ |
| Carter, Wm. W. | 69 W. 50th St., New York, N. Y., | ALR |
| Carvill, Lizzie M. | 101 Newbury St., Boston, Mass., | Op. |
| Cary, Edward H. | Dallas, Tex., | OALR |
| Cary, G. D. | Grand Junction, Colo. | |
| Case, Geo. M. | 154 N. Main St., Elmira, N. Y., | OALR |
| Castle, Chas. H. | The Poinciana, Avondale, Cincinnati, O., | OALR |
| Cavanaugh, John A. | 7 W. Madison St., Chicago, Ill., | ALR |
| Cayce, Eldred B. | 301-302 Hitchcock Bldg., Nashville, Tenn., | OALR |
| Chambers, Talbot R. | 15 Exchange Pl., Jersey City, N. J., | OALR |
| Chapman, Vernon A. | 1213 Wells Bldg., Milwaukee, Wis., | OALR |
| Charlton, C. Coulter. | 124 S. Illinois St., Atlantic City, N. J., | ALR |
| Cheatham, Wm. | 303 W. Chestnut St., Louisville, Ky., | OALR |
| Chenery, William E. | 222 Huntington Ave., Boston, Mass., | ALR |
| Cheney, F. E. | 126 Commonwealth Ave., Boston, Mass., | Op. |
| Childs, Hal A. | Creston, Ia., | OALR |
| Claiborne, John H., Jr. | 11 E. 48th St., New York, N. Y., | Op. |
| Clark, Frederick T. | 52 Broad St., Westfield, Mass., | OALR |
| Clark, Floyd H. | 337 Monroe Ave., Rochester, N. Y., | OALR |
| Clark, Howard S. | 616 Syndicate Block, Minneapolis, Minn., | OALR |
| Clark, John Sheldon. | Freeport, Ill., | OALR |
| Clement, Charles C. | 15 E. Washington St., Chicago, Ill., | OALR |
| Clevenger, W. F. | Newton Claypool Bldg., Indianapolis, Ind., | ALR |
| Cobb, Carolus Melville. | 10 Nahant St., Lynn, Mass., | OALR |
| Cobb, Edwin. | Masonic Temple, Marshalltown, Ia., | OALR |
| Coble, Paul Barnett. | 408 Penway Bldg., Indianapolis, Ind., | ALR ✓ |
| Cochran, Robert W. | Madison, Ind., | OALR |
| Cogan, Jas. E. | 707 Rose Bldg., Cleveland, O., | OALR |
| Coghlan, John. | 1104 Selling Bldg., Portland, Ore., | OALR |
| Cohen, Martin. | 1 W. 85th St., New York, N. Y., | OALR |
| Cohn, Felix. | 31 E. 60th St., New York, N. Y., | ALR |
| Cohn, Seldon. | Fulton, Ky., | OALR |
| Collins, E. W. | Metropolitan Bldg., Denver, Colo., | ALR |
| Collins, Homer. | New Jersey Bldg., Duluth, Minn., | OALR |
| Collins, Joseph Daniel. | 90 Main St., Northampton, Mass., | ALR ✓ |
| Colver, Benton H. | 315 Champion St., Battle Creek, Mich., | OALR |
| Conboy, Philip. | 82 East Ave., Rochester, N. Y., | OALR |
| Connor, Ray. | 703 Washington Arcade, Detroit, Mich., | OALR✓ |
| Cook, Sheldon E. | 504 Richards Block, Lincoln, Neb. | |
| Cooper, Claude E. | 646 Metropolitan Bldg., Denver, Colo. | |
| Coover, David H. | Metropolitan Bldg., Denver, Colo., | Op. |
| Corser, John B. | Scranton Private Hospital, Scranton, Pa., | OALR✓ |
| Corson, Geo. R. | 212 W. Market St., Pottsville, Pa., | OALR |

- Corwin, Arthur.....25 E. Washington St., Chicago, Ill., ALR
 Cott, Chester C.....1195 Main St., Buffalo, N. Y., ALR
 Cott, Geo. F.....1195 Main St., Buffalo, N. Y., ALR ▼
 Coulter, Charles F.....Wodena, Minn., OALR
 Covington, L. C.....1015 Virginia St., Charleston, W. Va., OALR ▼
 Cowper, H. W.....543 Franklin St., Buffalo, N. Y., Op.
 Craig, Alex. R.....535 N. Dearborn St., Chicago, Ill. ▼
 Crampton, George S.....1700 Walnut St., Philadelphia, Pa., Op. ▼
 Crary, A. W.....624½ Story St., Boone, Ia., OALR
 Crawford, James H.....Atlanta, Ga., OALR
 Creighton, W. J.....1905 Chestnut St., Philadelphia, Pa., Op.
 Creveling, H. C.....214-215 Humboldt Bldg., St. Louis, Mo., ALR
 Crisp, Wm. Henry.....318 Majestic Bldg., Denver, Colo., Op.
 Crockett, Robert L.....Oneida, N. Y.
 Croskey, J. W.....3325 Powelton Ave., Philadelphia, Pa., OALR
 Cross, Albert Elmer.....1045 Slater Bldg., Worcester, Mass., Op.
 Crossfield, Frederick.....Hartford, Conn., ALR
 Culbertson, L. R.....Masonic Temple, Zanesville, O., OALR
 Cullom, Marvin M.....Hitchcock Bldg., Nashville, Tenn., OALR
 Curdy, Robert J.....Rialto Bldg., Kansas City, Mo.
 Curry, Glendon E.....Westinghouse Bldg., Pittsburgh, Pa., Op.
 Curtis, Wesley L.....Fairbury, Neb., OALR
 Cutler, Franklin E.....936 Rose Bldg., Cleveland, O., ALR ▼

 Dabney, S. G.....216 W. Chestnut, Louisville, Ky., OALR
 Dabney, Virginius.....1633 Connecticut St., Washington, D. C., ALR
 Dabney, William R.....Cuyahoga Falls, O.
 Dames, A. F.....Metropolitan Bldg., St. Louis, Mo., ALR
 Dancey, A. B.....First Natl. Bank Bldg., Jackson, Tenn., OALR
 Davies, T. Alexander.....578 Sherbourne St., Toronto, Can., OALR
 Davis, A. E.....50 W. 37th St., New York, N. Y., Op.
 Davis, Charles H.....513 Walnut St., Knoxville, Tenn., OALR
 Davis, G. E.....50 W. 37th St., New York, N. Y., OALR
 Davis, J. Leslie.....1700 Walnut St., Philadelphia, Pa., ALR
 Davis, James Edward.....McAlister, Okla.
 Day, E. W.....Westinghouse Bldg., Pittsburgh, Pa., ALR ▼
 Dayton, Glenn Orville.....403 Lewisohn Bldg., Butte, Mont., OALR
 Dayton, W. L.....302 Funke Bldg., Lincoln, Neb., OALR
 De Lue, Frederick Spaulding.....99 Newbury St., Boston, Mass., Op.
 Dean, F. W.....401 City Natl. Bank Bldg., Council Bluffs, Ia., OALR
 Dean, L. W.....12 S. Clinton St., Iowa City, Ia., OALR
 Dean, Stanley Smith.....LaCrosse, Wis.
 Decherd, Henry B.....Dallas, Tex., OALR
 Decker, J. C.....Cheyenne, Wyo.
 Dennis, D. N.....221 W. 9th St., Erie, Pa., Op.A
 Dennis, Frank L.....501 N. Tijon St., Colorado Springs, Colo., ALR ▼
 Dennis, Geo. J.....25 E. Washington St., Chicago, Ill., ALR
 Derby, George S.....7 Hereford St., Boston, Mass., Op. ▼
 Dernehl, Paul Herman.....717 Majestic Bldg., Milwaukee, Wis., Op.
 Detling, Frank Edward.....1112 Title Ins. Bldg., Los Angeles, Calif., OALR
 Dewey, Christian H.....U. S. Indian Office, Washington, D.C., Op.
 DeVilbiss, Allen.....1009 Lincoln Ave., Toledo, O., L
 Dickinson, B. M.....Keenan Bldg., Pittsburgh, Pa., ALR
 Dietrich, W. A.....404-405 Temple Court, Chattanooga, Tenn., OALR
 Dillon, John A.....310 Main St., Springfield, Mass., ALR
 Dimitry, T. J.....Maison Blanche Bldg., New Orleans, La., Op.
 Dixon, Geo. Sloan.....40 E. 41st St., New York, N. Y., R
 Dixon, W. Eugene.....
706-8 State Bank Bldg., Oklahoma City, Okla.
 Doane, L. Leo.....I. O. O. F. Temple, Butler, Pa., OALR
 Dodd, Oscar.....30 N. Michigan Blvd., Chicago, Ill., Op.A
 Donohoe, Wm. D.....829 Boston Bldg., Salt Lake City, Utah
 Donovan, J. A.....Phoenix Bldg., Butte, Mont., OALR
 Dorento, Dred R.....Merchants Natl. Bank Bldg., Fort Smith, Ark., OALR

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| Dorsey, J. G. | 105 Douglas Ave., Wichita, Kan., | OALR |
| Dow, Frank Edward | Central Chambers, Northampton, Mass., | Op. |
| Dowling, J. I. | 116 Washington Ave., Albany, N. Y., | OALR |
| Dowling, J. Thomas | 415 Cobb Bldg., Seattle, Wash. | |
| Dowling, Oscar | First Natl. Bank Bldg., Shreveport, La., | OALR |
| Dresel, Gustav | 209 Post St., San Francisco, Calif. | |
| Duane, Alexander | 139 East 37th St., New York, N. Y., | Op. |
| Duane, Joseph F. | Peoria, Ill., | OALR |
| Dudley, Wm. H. | 512 Boeckman Bldg., Los Angeles, Calif., | OALR |
| Duncan, Adelaide | 32 N. State St., Chicago, Ill., | OALR |
| Dunn, Ira J. | Masonic Temple, Erie, Pa., | OALR |
| Dunn, Murison | Richmond, Ky., | OALR |
| Dunn, Oscar B. | Fourth and Railroad Sts., Ironton, O. | |
| Duntley, George S. | Bushnell, Ill., | OALR |
| Dutrow, Howard V. | 922 Reibold Bldg., Dayton, O., | OALR |
| Dwight, Corydon G. | Madison, Wis., | OALR |
| Dworetzky, Julius | New York City Sanitarium, Otisville, N. Y., | ALR |
| Earel, Albert M. | Hoopeston, Ill., | OALR |
| Earel, John Wm. | Sterns Bldg., Quincy, Ill., | OALR |
| Eastabrook, Charles | 390 Main St., Worcester, Mass., | OALR |
| Eastman, Henry | 1104 Park Bldg., Pittsburgh, Pa., | Op. |
| Easton, John C. | 84 High St., Springfield, O., | OALR |
| Edgar, Thomas O. | Dixon, Ill., | OALR |
| Edson, Ray | 560 Delaware Ave., Buffalo, N. Y., | Op. |
| Edwards, C. J. | Vicksburg, Miss., | OALR |
| Edwards, Stocomb R. | 205-7 Widdicomb Bldg., Grand Rapids, Mich. | |
| Edwards, William A. | 329 Main St., La Crosse, Wis., | OALR |
| Eigler, C. O. | Metropolitan Bldg., Denver, Colo. | |
| Ellegood, Joshua A. | Equitable Bldg., Wilmington, Del., | OALR |
| Ellett, Edward C. | Exchange Bldg., Memphis, Tenn., | Op. |
| Ellis, James C. | Houston, Tex., | OALR |
| Emerson, Linn | 105 W. 50th St., New York, N. Y., | OALR |
| Engle, H. P. | 200 Olive St., Newton, Ia., | OALR |
| Esterly, D. E. | 813 Kansas Ave., Topeka, Kan., | OALR |
| Evensen, Harold O. | Ottawa, Ill., | OALR |
| Ewing, U. B. G. | Knollenberg Annex Bldg., Richmond, Ind., | OALR |
| Fagin, Robert | Exchange Bldg., Memphis, Tenn., | Op. |
| Fairbairn, John F. | 131 Allen St., Buffalo, N. Y., | ALR |
| Faith, Thomas | 31 N. State St., Chicago, Ill., | OALR |
| Fallows, Howard D. | New Hampton, Ia. | |
| Farmer, Alfred G. | 5 Park Pl., Athens, O. | |
| Farrell, P. J. H. | 25 E. Washington St., Chicago, Ill., | OALR |
| Farrington, Pope M. | Bank of Commerce and Trust Bldg., Memphis, Tenn., | ALR |
| Fawcett, Ivan | 1307 Chapline St., Wheeling, W. Va., | OALR |
| Feingold, Marcus | 503 Medical Bldg., New Orleans, La., | Op. |
| Fellows, G. Gurnee | 30 N. Michigan Blvd., Chicago, Ill., | OALR |
| Felt, Carl L. | 2007 Chestnut St., Philadelphia, Pa. | |
| Ferguson, Edmund S. | State Natl. Bank Bldg., Oklahoma City, Okla., | OALR |
| Fernandez, Francisco M. | Prado 105, Havana, Cuba, | OALR |
| Ferris, Edgar | Newcastle, Ind., | OALR |
| Finnoff, Wm. C. | 520 Metropolitan Bldg., Denver, Colo., | Op.A |
| Fish, H. Manning | 29 E. Madison St., Chicago, Ill. | |
| Fisher, Carl | Rochester, Minn., | Op.A |
| Fisher, Mark Ohl | Parkersburg, W. Va., | OALR |
| Fisher, Valentine | 202 Mercantile Bank Bldg., Boulder, Colo. | |
| Fisher, Wm. A. | 31 N. State St., Chicago, Ill., | OALR |
| Fiske, Geo. F. | 25 E. Washington St., Chicago, Ill., | OALR |
| Fitzpatrick, T. V. | 19 W. 7th St., Cincinnati, O., | ALR |
| Flagg, John D. | 473 Virginia St., Buffalo, N. Y., | Op.A |

- Flatley, Thomas Joseph.....206 Reliance Bldg., Moline, Ill., OALR
 Fleming, E. W....Trust and Savings Bldg., Los Angeles, Calif., ALR
 Fletcher, John Rice.....25 E. Washington St., Chicago, Ill., ALR
 Floyd, Thomas W.....Peoria, Ill., OALR
 Foley, John Wm.....217 Majestic Bldg., Denver, Colo., OALR
 Forbes, Henry H.....40 E. 41st St., New York, N. Y., ALR ▼
 Forsyth, Edgar A.....322 Franklin St., Buffalo, N. Y., ALR
 Foster, E. Edwin.....271 Union St., New Bedford, Mass., OALR
 Foster, Hal.....11th and Walnut, Kansas City, Mo., ALR
 Foster, John H.....405 Carter Bldg., Houston, Tex., ALR
 Foster, John McEwen.....11 Stedman Bldg., Denver, Colo., OALR
 Fowler, Samuel R.....918 University Bldg., Syracuse, N. Y., ALR
 Fox, L. Webster.....1636 Spruce St., Philadelphia, Pa.
 Fox, Ralph D.....411-13 Unity Bldg., Bloomington, Ill., OALR
 Francis, Charles Horace....30 N. Michigan Blvd., Chicago, Ill., Op.
 Francis, Lee Masten.....636 Delaware Ave., Buffalo, N. Y., Op. ▼
 Frank, Ira.....104 S. Michigan Blvd., Chicago, Ill., ALR
 Franklin, W. S.....Butler Bldg., San Francisco, Calif., Op.
 Frederick, M. W.....135 Stockton St., San Francisco, Calif., OALR
 Freedman, L. M.....419 Boylston St., Boston, Mass., ALR
 French, Royal F.....Center and Main Sts., Marshalltown, Ia., OALR
 Freudenthal, Wolff.....59 E. 75th St., New York, N. Y., ALR
 Frey, C. L.....Dime Bank Bldg., Scranton, Pa., OALR
 Fridenberg, Percy H.....38 W. 59th St., New York, N. Y., OALR
 Friedman, David.....808 West End Ave., New York, N. Y., ALR
 Friendenwald, Harry.....1522 Madison Ave., Baltimore, Md., Op.A
 Fringer, W. R.....William Brown Bldg., Rockford, Ill., Op.
 Frost, W. S.....507 Paulsen Bldg., Spokane, Wash., OALR ▼
 Fryer, John L.....Leavenworth, Kan., OALR
 Fulkerson, Clarke B.....
308 Kalamazoo Natl. Bank Bldg., Kalamazoo, Mich., OALR
 Fullenwider, Charles M....405 Barnes Bldg., Muskogee, Okla., OALR
 Fuller, Theron E.....State Bank Bldg., Texarkana, Ark., OALR ▼
 Furgason, Allen P.....303 Richards Bldg., Lincoln, Neb.
 Gale, J.....Denver, Colo.
 Gallaher, T. J.....605 California Bldg., Denver, Colo., ALR
 Garber, Jerry M.....King Bldg., Mansfield, O., OALR
 Garraghan, E. F.....31 N. State St., Chicago, Ill., OALR
 Garrett, John R.....Strickland Bldg., Roanoke, Va., OALR
 Gatewood, Wm. Lawrence....15 E. 48th St., New York, N. Y., ALR
 Gavin, Edward F.....Waukegan, Ill., OALR
 Geiger, C. W.....269½ Schuyler St., Kankakee, Ill., OALR
 George, Henry W.....254 Spring St., Middletown, Pa.
 Gibson, R. D..Dollar Savings and Trust Bldg., Youngstown, O., OALR
 Gifford, Harold.....Brandeis Bldg., Omaha, Neb., Op.A
 Gilbert, Frank Y.....148 Park St., Portland, Me. ▼
 Gill, Geo.....Elyria, O.
 Gill, Michael H.....Hartford, Conn., Op.A
 Gillman, Robert W.....107 W. Fort St., Detroit, Mich., Op.
 Givens, L. S....Harrison Deposit Bank Bldg., Cynthiaana, Ky., OALR
 Gleason, E. B.....2033 Chestnut St., Philadelphia, Pa., ALR ▼
 Glogau, Otto.....132 Madison Ave., New York, N. Y., OALR
 Glosser, Herbert.....404 Franklin St., Buffalo, N. Y., OALR
 Glover, Samuel P.....1118, 12th Ave., Altoona, Pa.
 Goddard, H. M.....1338 Spruce St., Philadelphia, Pa., ALR
 Goldbach, Leo John.....322 N. Charles St., Baltimore, Md., OALR
 Goldsmith, Perry.....84 Carlton St., Toronto, Canada
 Goldstein, M. A.....3858 Westminster Pl., St. Louis, Mo., ALR ▼
 Good, R. H.....7 W. Madison St., Chicago, Ill., OALR
 Goodell, William.....6 Chestnut St., Springfield, Mass., ALR ▼
 Gorrell, Talbot J.....93 Illinois St., Chicago Heights, Ill., OALR
 Gourley, Geo. T.....Sinclair Bldg., Stubenville, O.
 Goux, L. J.....545 David Whitney Bldg., Detroit, Mich., OALR

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| Gradle, Harry S. | 32 N. State St., Chicago, Ill., | Op. ▼ |
| Graham, H. B. | 209 Post St., San Francisco, Calif., | ALR |
| Graham, Richard W. | 632 Consolidated Realty Bldg., Los Angeles, Calif. | |
| Grant, F. E. | Kalamazoo Natl. Bank, Kalamazoo, Mich., | OALR |
| Grant, J. George. | Woods Block, Akron, O., | Op. |
| Graves, Wilbur H. | Pittsburg, Kan., | OALR |
| Green, Aaron S. | 210 Post St., San Francisco, Calif. | |
| Green, John, Jr. | 625 Metropolitan Bldg., St. Louis, Mo., | Op. |
| Green, Louis D. | 210 Post St., San Francisco, Calif. | |
| Greenwood, Allen. | 101 Newbury St., Boston, Mass., | Op. ▼ |
| Grim, Ulysses Joshua. | 31 N. State St., Chicago, Ill., | OALR |
| Grosvenor, L. N. | The Medical Block, Huron, S. D., | OALR |
| Grove, Robt. K. | 239 Delaware Ave., Buffalo, N. Y., | OALR |
| Grove, Wm. E. | Wells Bldg., Milwaukee, Wis., | OALR |
| Grushlow, Israel. | 271 Central Park, W., New York, N. Y., | ALR ▼ |
| Guggenheim, Louis K. | 726 Metropolitan Bldg., St. Louis, Mo., | ALR |
| Gundelach, C. Armin. | 4500 Olive St., St. Louis, Mo., | ALR |
| Guthrie, A. L. | Indian Bldg., Oklahoma City, Okla., | OALR ▼ |
| Guthrie, James M. | Meridian, Miss., | OALR ▼ |
| Guttman, John. | 60 St. Marks Pl., New York, N. Y., | OALR |
| Guyton, B. S. | University, Miss. | |
| Gwinn, G. E. | San Antonio, Tex., | OALR |
| Haden, Henry C. | 203 Trust Bldg., Galveston, Tex., | OALR |
| Hadley, James William. | Frankfort, Ind., | OALR |
| Hager, Walter A. | 103 N. Lafayette St., South Bend, Ind., | OALR |
| Hagler, A. L. | 410 E. Capitol Ave., Springfield, Ill., | OALR |
| Hagler, Elmer E. | 410 E. Capitol Ave., Springfield, Ill., | OALR |
| Haley, Peter A. | Charlestown, W. Va., | OALR |
| Hall, G. C. | Gaulbert Bldg., Louisville, Ky., | OALR |
| Halstead, Fred. S. | 708 Metropolitan Bldg., Denver, Colo. | |
| Hampton, Robt. R. | 804 Boston Bldg., Salt Lake City, Utah, | OALR ▼ |
| Hancock, James Cole. | 135 Cambridge Pl., Brooklyn, N. Y., | Op. |
| Hardy, Wm. F. | Metropolitan Bldg., St. Louis, Mo., | Op. |
| Harkness, Gordon F. | Third and Brady Sts., Davenport, Ia., | OALR ▼ |
| Harris, Clarence. | Johnstown Trust Bldg., Johnstown, Pa., | OALR |
| Harris, H. B. | Reibold Bldg., Dayton, O., | OALR |
| Harris, W. C. | Groton Bldg., Cincinnati, O., | ALR |
| Harrison, W. G. | Birmingham, Ala., | OALR |
| Hartley, F. A. | Springfield, O., | OALR |
| Hartz, H. J. | 27 E. Adams St., Detroit, Mich., | ALR |
| Hartzell, Sol. M. | Dollar Bank Bldg., Youngstown, O., | OALR |
| Harvey, N. D. | 114 Waterman St., Providence, R. I., | OALR |
| Haskell, Alfred D. | 145 High St., Portland, Me., | Op. ▼ |
| Haskin, Wm. Henry. | 40 E. 41st St., New York, N. Y., | ALR ▼ |
| Haspel, M. David. | 124 Baronne St., New Orleans, La., | ALR |
| Hatch, C. B. | 3 W. Church St., Newark, O., | OALR |
| Hauer, Arthur Merle. | 246 E. State St., Columbus, O., | OALR ▼ |
| Haughey, Wilfrid. | 24 W. Main St., Battle Creek, Mich., | OALR ▼ |
| Hawley, Alanson. | Seattle, Wash. | |
| Hawley, Clark W. | 7 W. Madison St., Chicago, Ill., | OALR |
| Hayden, A. A. | 32 N. State St., Chicago, Ill., | OALR ▼ |
| Hayman, Edward Chapman. | Lincoln, Neb., | OALR |
| Hays, Harold N. | 11 W. 81st St., New York, N. Y., | ALR |
| Hays, Samuel B. | Atherton Bldg., Louisville, Ky., | OALR |
| Hazlett, Leslie R. | 106 W. Diamond St., Butler, Pa., | OALR |
| Heard, C. F. | Erie, Pa., | ALR |
| Heard, Mary Katherine. | 24 N. Clinton St., Iowa City, Ia., | Op. |
| Heath, Albert C. | Lowry Bldg., St. Paul, Minn., | ALR |
| Heath, Frederick Carroll. | | |
| | Newton Claypool Bldg., Indianapolis, Ind., | Op. |
| Heckel, E. B. | Jenkins Bldg., Pittsburgh, Pa., | Op. |
| Heckler, Frank A. | 106 E. Broad St., Columbus, O., | OALR |

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| Heeb, Harry J. | 8-9 Loan and Trust Bldg., Milwaukee, Wis., | OALR |
| Heed, Chas. Rittenhouse | 1402 Spruce St., Philadelphia, Pa., | Op. |
| Heflebower, R. C. | 22 W. 7th St., Cincinnati, O., | OALR |
| Heggie, Norman M. | Buckman Bldg., Jacksonville, Fla., | OALR |
| Heitger, Joseph Daniel | | |
| | Atherton Bldg., 608 Fourth St., Louisville, Ky., | OALR |
| Henderson, Frank L. | Humboldt Bldg., St. Louis, Mo., | Op. |
| Hénning, Carl | The Rochambeau, Washington, D. C., | Op. |
| Henninger, L. L. | Council Bluffs, Ia., | OALR |
| Herriman, L. L. | Alamasa, Colo., | |
| Hess, Wm. L. | 400 California Bldg., Denver, Colo., | OALR |
| Hetzel, Clarence Charles | 512 Eccles Bldg., Ogden, Utah, | OALR |
| Hickey, Preston M. | 32 W. Adams St., Detroit, Mich., | R |
| Higbee, Edward H. | 417 Metropolitan Bldg., St. Louis, Mo., | Op |
| Higginbotham, Thomas L. | | |
| | First Natl. Bank Bldg., Hutchinson, Kan., | OALR |
| Higgins, Samuel George | 213 Wells Bldg., Milwaukee, Wis., | OALR |
| Hill, Arthur J. | 335 S. Cleveland Ave., Canton, O., | OALR |
| Hill, Emory | 30 N. Michigan Blvd., Chicago, Ill., | Op. |
| Hill, J. Frederick | Waterville, Me., | OALR |
| Hilger, A. W. | 925 Lowry Bldg., St. Paul, Minn., | ALR |
| Hinnen, G. A. | 8-10 E. 8th St., Cincinnati, O., | OALR |
| Hitz, H. B. | Goldsmith Bldg., Milwaukee, Wis., | ALR |
| Hockney, Francis J. | Chattanooga, Tenn., | Op.AL |
| Hodsdon, B. F. | Miami, Fla., | OALR |
| Hoffman, J. R. | 31 N. State St., Chicago, Ill., | OALR |
| Hogshead, J. M. | 602 Georgia Ave., Chattanooga, Tenn., | OALR |
| Hogue, Gustavus I. | 410 Jefferson St., Milwaukee, Wis., | OALR |
| Hogue, Delso W. | Fairbanks Bldg., Springfield, O., | OALR |
| Hohf, Julius | Yankton, S. D., | OALR |
| Holloway, Thomas B. | 1819 Chestnut St., Philadelphia, Pa., | Op. |
| Holmes, Christian R. | 8-10 E. 8th St., Cincinnati, O., | OALR |
| Holmes, Edgar M. | 531 Beacon St., Boston, Mass., | ALR |
| Holt, Erastus E. | 723 Congress St., Portland, Me., | Op.A |
| Holt, Erastus E., Jr. | 723 Congress St., Portland, Me., | Op.A |
| Hompes, Joseph J. | Lincoln, Neb., | OALR |
| Hood, Thomas C. | 224 N. Meridian St., Indianapolis, Ind., | Op |
| Hoode, Anders G. | Board of Trades Bldg., Superior, Wis. | |
| Hooker, Rufus W. | | |
| | 1240 Bank of Commerce Bldg., Memphis, Tenn., | OALR |
| Hopkins, W. E. | 515 Park Ave., New York, N. Y., | OALR |
| Horn, Henry | 209 Post St., San Francisco, Calif., | ALR |
| Howard, Joseph Wm. | 910 Rialto Bldg., Kansas City, Mo., | OALR |
| Howard, Wm. L. | 11th Floor, Exchange Bldg., Memphis, Tenn., | OALR |
| Howe, Lucien | 522 Delaware Ave., Buffalo, N. Y., | Op. |
| Hubbard, Albert E. | 372 Franklin St., Buffalo, N. Y., | Op. |
| Hubby, Lester M. | 27 W. 68th St., New York, N. Y., | ALR |
| Huffman, John R. | 1231 Greenwood Ave., Wilmette, Ill. | |
| Hughes, William F. | | |
| | 327 Newton Claypool Bldg., Indianapolis, Ind., | Op. |
| Huizinga, J. G. | Grand Rapids, Mich., | OALR |
| Hunt, H. E. | Lowry Bldg., St. Paul, Minn., | OALR |
| Hunter, D. W. | 80 W. 40th St., New York, N. Y., | Op. |
| Hunter, John Emerson | Greenville, O., | OALR |
| Hurd, Lee M. | 15 E. 48th St., New York, N. Y., | ALR |
| Hurley, Edward Daniel | 419 Boylston St., Boston, Mass., | Op. |
| Hussey, Edward John | 276 High St., Holyoke, Mass., | Op.LR |
| Iglauer, Samuel | 22 W. 7th St., Cincinnati, O., | ALR |
| Imperator, Charles J. | 245 W. 102d St., New York, N. Y., | ALR |
| Ingerman, Serguis M. | 1843 Madison Ave., New York, N. Y., | OALR |
| Ingersoll, J. M. | 1021 Prospect Ave., Cleveland, O., | ALR |
| Inglis, Harry James | 483 Beacon St., Boston, Mass., | ALR |
| Ingram, Lawrence C. | DeLand, Fla., | OALR |

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| Irvine, Alex. R. | Kearns Bldg., Salt Lake City, Utah | ▼ |
| Irwin, Frank Newton | 527, 5th Ave., New York, N. Y. | OALR |
| Irwin, Vincent J. | 351 Main St., Springfield, Mass. | OALR |
| Isaacs, David | 816 Brandeis Theater Bldg., Omaha, Neb. | OALR |
| Israel, Sidney | 404 Carter Bldg., Houston, Tex. | OALR |
| Ivins, Harry M. | 408 Second Ave., Cedar Rapids, Ia. | OALR |
| Jacobs, Max W. | 606 Carleton Bldg., St. Louis, Mo. | Op. ▼ |
| Jackson, Chevalier | 261 S. 17th St., Philadelphia, Pa. | |
| Jackson, Edward | Majestic Bldg., Denver, Colo. | Op. |
| Jackson, F. A. | Salida, Colo. | |
| Jacobi, Frank | 416-418 Colton Bldg., Toledo, O. | OALR |
| Jacobs, Simon M. | 1187 Boston Rd., New York, N. Y. | OALR |
| Jacoby, Douglas P. A. | Newport, R. I. | Op.AL |
| James, John A. J. | 1006 Carleton Bldg., St. Louis, Mo. | ALR |
| James, John H. | Natl. Citizens Bank Bldg., Mankato, Minn. | OALR |
| Jamison, T. H. | Wellington, Kan. | OALR |
| Jarecky, Herman | 138 W. 86th St., New York, N. Y. | OALR |
| Jennings, J. E. | 509 Carleton Bldg., St. Louis, Mo. | OALR |
| Jessaman, Leon Webster | 30 Hollis St., Framingham, Mass. | OALR ▼ |
| Jobson, G. B. | Franklin, Pa. | OALR |
| Johnson, Anders E. | Watertown, S. D. | OALR |
| Johnson, H. R. | 512 Walnut Ave., Fairmont, W. Va. | OALR |
| Johnson, J. H. | 106 W. 8th St., Coffeyville, Kan. | OALR |
| Johnson, Otis H. | 55 W. 95th St., New York, N. Y. | OALR ▼ |
| Johnston, Richard Hall | 807 N. Charles St., Baltimore, Md. | |
| Johnston, William H. | Hershey Bldg., Muscatine, Ia. | OALR |
| Johnston, Wilson | Portland, Ore. | ▼ |
| Jones, Elgin W. | 44 Atlantic St., Lynn, Mass. | Op.A |
| Jones, Emmett Lee | Cumberland, Md. | OALR |
| Jones, Newbold C. | 42 Prince Arthur Ave., Toronto, Can. | OALR |
| Joyes, Crittenden | | |
| Farmers' and Mechanics' Natl. Bank Bldg. | Fort Worth, Tex. | |
| Julien, Wm. Floyd | 432 Madison St., Gary, Ind. | OALR |
| Kaufman, A. Spencer | 1633 Spruce St., Philadelphia, Pa. | ALR |
| Keeler, J. Clarence | 4059 Spruce St., Philadelphia, Pa. | ALR ▼ |
| Keiper, Geo. F. | Lafayette, Ind. | OALR |
| Keith, Darwin M. | 414 N. Main St., Rockford, Ill. | ALR |
| Keith, Willis E. | Excelsior Springs, Mo. | OALR |
| Keller, W. S. | 7th and Race Sts., Cincinnati, O. | OALR |
| Keller, T. F. | The Spitzer, Toledo, O. | OALR |
| Kelley, S. G. | Sedalia, Mo. | |
| Kelly, C. W., Jr. | Atherton Bldg., Louisville, Ky. | OALR |
| Kerrison, P. D. | 58 W. 56th St., New York, N. Y. | ALR |
| Key, Benn Witt | 7 W. 49th St., New York, N. Y. | Op. |
| Kiefer, Hugo A. | 406 Brockman Bldg., Los Angeles, Calif. | |
| Kimball, A. H. | The Farragut (17th and I Sts.), Washington, D. C. | Op.AL |
| Kimberlin, Joseph W. | 900 Rialto Bldg., Kansas City, Kan. | Op. ▼ |
| Kincaid, John H. | Ross Flats, Knoxville, Tenn. | OALR |
| King, Clarence | 1801 Union Central Bldg., Cincinnati, O. | Op. ▼ |
| King, Geo. L. | 537 E. Market St., Alliance, O. | OALR |
| King, James J. | 40 E. 41st St., New York, N. Y. | ALR |
| King, Ossian H. | Hot Springs, Ark. | OALR |
| Kirkendall, John S. | Ithaca, N. Y. | Op.A |
| Kirkpatrick, Samuel | Lauderdale St., Selma, Ala. | OALR |
| Kiser, J. D. | 28 Aylesford Pl., Lexington, Ky. | OALR |
| Kistler, Oliver F. | 43 N. Franklin St., Wilkes-Barre, Pa. | Op.AR |
| Kistner, Frank B. | 909 Stevens Bldg., Portland, Ore. | ALR |
| Kittelston, Theodore | Fergus Falls, Minn. | OALR |
| Kleene, Frederick | 801 Milwaukee Ave., Chicago, Ill. | OALR |
| Kline, Harold G. | 902 University Block, Syracuse, N. Y. | ALR |
| Klinedinst, J. Fred | 220 S. George St., York, Pa. | OALR |

- Knapp, Arnold.....10 E. 54th St., New York, N. Y., Op.
 Knapp, A. J.....Intermediate Life Bldg., Evansville, Ind., OALR
 Knapp, Bleeker.....Evansville, Ind., OALR
 Knapp, Henry Clay.....Huntingburg, Ind., OALR
 Kneedler, G. C.....Jenkins Bldg., Pittsburgh, Pa., ALR
 Knode, A. R.....Omaha Natl. Bank Bldg., Omaha, Neb., ALR
 Knowles, Wm. Fletcher.....220 Clarendon St., Boston, Mass., ALR ▼
 Knox, James McBryde.....Cedar Rapids, Ia., OALR
 Kollock, Chas. Wilson.....Charleston, S. C., OALR ▼
 Kopetzky, S. J.....616 Madison Ave., New York, N. Y., ALR ▼
 Kraft, Oscar H.....108 N. State St., Chicago, Ill., Op.
 Krauss, Frederick.....1701 Chestnut St., Philadelphia, Pa., OALR
 Krebs, Adolph.....7040 Jenkins Arcade, Pittsburgh, Pa., Op.
 Kress, Geo. H.....Bradbury Bldg., Los Angeles, Calif.
 Kreutzer, Alfred G.....135 Grand Ave., Milwaukee, Wis., ALR
 Krieger, Geo. M.....4800 Whetsel Ave., Station M., Cincinnati, O., OALR ▼
 Krug, E. F.....12 W. 44th St., New York, N. Y., OALR ▼
 Kunz, Hugo.....2 W. 123d St., New York, N. Y., ALR
 Kyle, J. J.....Suite 702, Title Insurance Bldg., Los Angeles, Calif., OALR
- LaForce, Bert D.....Ottumwa, Ia., OALR
 LaForce, E. F.....Tama Bldg., Burlington, Ia., OALR
 Lamb, F. W.....24 E. 8th St., Cincinnati, O., OALR
 Lamb, Robert S.....Stoneleigh Court, Washington, D. C., Op.
 Lancaster, Walter B.....522 Commonwealth Ave., Boston, Mass., Op. ▼
 Lance, Arthur J.....Portsmouth, N. H., OALR
 Landfried, Charles J.....Perrin Bldg., New Orleans, La., ALR
 Landman, Louis H.....14 E. 7th St., Cincinnati, O., OALR
 Langworthy, Henry Glover...10th and Bluff Sts., Dubuque, Ia., OALR
 Laning, J. Halcombe.....Commerce Bldg., Kansas City, Mo.
 Lapsley, R. M.....100 N. 5th St., Keokuk, Ia., OALR
 Large, S. H.....Rose Bldg., Cleveland, O., ALR
 Larkin, Bernard J.....514 Hume-Mansur Bldg., Indianapolis, Ind., Op. ▼
 Larsen, Carl.....Lowry Bldg., St. Paul, Minn., OALR
 Lassen, Fritz.....First Natl. Bank Bldg., Pueblo, Colo., ALR
 Lauder, Edward.....1021 Prospect St., Cleveland, O., Op.
 Lauder, Clark Hays.....Grinnell, Ia., OALR ▼
 LaVigne, Alexander A.....129 W. 46th St., New York, N. Y., OALR
 Lawrence, Granville.....1212 Spruce St., Philadelphia, Pa., Op.
 Lawrence, N. Louise.....513 W. St. Catharine St., Louisville, Ky., ALR
 Layman, D. W.....Hume-Mansur Bldg., Indianapolis, Ind., ALR
 Leavy, Charles A.....426 Metropolitan Bldg., St. Louis, Mo., ALR ▼
 Ledbetter, S. L.....First Natl. Bank Bldg., Birmingham, Ala., OALR
 Lederman, Isaac.....Atherton Bldg., Louisville, Ky., OALR
 Leech, James Wm.....111 Broad St., Providence, R. I., OALR
 Lefler, Anna B.....886 Kensington Rd., Los Angeles, Calif.
 Lehan, James W.....Greeley, Colo., OALR
 Lenker, John N.....Osborn Bldg., Cleveland, O., ALR
 Lemere, Henry Bassett.....
400 Brandeis Theater Bldg., Omaha, Neb., OALR ▼
 Lent, Edwin J.....South Bend, Ind., OALR
 Lester, Harry S.....219 E. Main St., Streator, Ill., OALR ▼
 Levi, Walter Dean.....Starks Bldg., Louisville, Ky., OALR
 Levy, Louis.....931 Bank of Commerce Bldg., Memphis, Tenn., OALR
 Levy, Robert.....Metropolitan Bldg., Denver, Colo., ALR ▼
 Lewis, Archibald C.....Memphis, Tenn., OALR
 Lewis, F. Park.....454 Franklin St., Buffalo, N. Y., Op.
 Lewis, G. Griffin.....University Block, Syracuse, N. Y., OALR
 Lewis, James Prince.....55 Grant St., Waltham, Mass., ALR
 Lewis, R. H.....5 W. Hargett St., Raleigh, N. C., Op.
 Lewitt, Frederick Clinton...291 Geary St., San Francisco, Calif., OALR ▼
 Libby, Geo. F.....Metropolitan Bldg., Denver, Colo.
 Lichtenberg, J. S.....819 Rialto Bldg., Kansas City, Mo., Op. ▼
 Lindsay, Geo.....Brittania Bldg., West St., Durban, Africa

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| Lindsey, David M. | 808 Boston Bldg., Salt Lake City, Utah, | OALR |
| Linhart, Christopher P. | 106 E. Broad St., Columbus, O., | OALR |
| Linn, Ellis G. | Des Moines, Ia., | OALR |
| Lintleman, Fred. R. | 312 Hayes Bldg., Jonesville, Wis. | |
| Lischkoff, Mozart | 366-370 Brent Bldg., Pensacola, Fla., | OALR▼ |
| Little, Albion Henry | 763 Congress St., Portland, Me., | Op. ▼ |
| Lloyd, Alijah W. | Hammond, Ind., | OALR▼ |
| Lockard, L. B. | Metropolitan Bldg., Denver, Colo., | ALR |
| Loeb, Clarence | 25 E. Washington St., Chicago, Ill., | Op. |
| Loeb, Hanau W. | Humboldt Bldg., St. Louis, Mo., | ALR▼ |
| Lokey, H. M. | 833 Candler Bldg., Atlanta, Ga., | OALR |
| Loomis, Earl A. | 204 Donaldson Bldg., Minneapolis, Minn. | |
| Lorie, Alvin | 409 Commerce Bldg., Kansas City, Mo. | |
| Loring, J. B. | 31 N. State St., Chicago, Ill., | OALR |
| Louchery, Daniel C. | 212 W. Main St., Clarksburg, W. Va., | OALR |
| Love, J. King | 42 N. 2d St., Easton, Pa., | Op. |
| Love, Louis F. | 1305 Locust St., Philadelphia, Pa., | Op. |
| Lowell, W. Holbrook | 101 Newbury St., Boston, Mass., | Op. |
| Luedde, W. H. | 311 Metropolitan Bldg., St. Louis, Mo., | Op. ▼ |
| Lukens, Chas. | 218 Michigan St., Toledo, O., | OALR |
| Lutz, Stephen Henry | 284 Hancock St., Brooklyn, N. Y., | ALR |
| Lynah, Henry L. | 127 W. 58th St., New York, N. Y. | |
| Lynch, Robert C. | 624 Gravier St., New Orleans, La., | ALR |
| Lyle, William | 300 Harrison Bldg., Augusta, Ga., | OALR▼ |
| Macfarlan, Douglas | 1805 Chestnut St., Philadelphia, Pa., | OALR |
| Macfarlane, Percy B. | 152 James St., South Hamilton, Ont., | OALR |
| MacKenzie, George | 1831 Chestnut St., Philadelphia, Pa., | OALR |
| MacLachlan, Archibald A. | 1111 Empire Bldg., Pittsburgh, Pa., | ALR ▼ |
| Madden, P. R. | Xenia, O., | OALR |
| Magee, Robert S. | Mills Bldg., Topeka, Kan., | Op.A |
| Magruder, Alexander C. | | |
| | 323 Burns Bldg., Colorado Springs, Colo., | OALR▼ |
| Maire, L. E. | 203 Park Bldg., Detroit, Mich., | Op.A |
| Mann, R. H. T. | Texarkana, Tex., | OALR |
| Mansur, D. W. | 1109 Brockman Bldg., Los Angeles, Calif. | |
| Markel, J. Clyde | 1005 Westinghouse Bldg., Pittsburgh, Pa. | |
| Marshall, Augustus L. | | |
| | 520 Hume-Mansur Bldg., Indianapolis, Ind., | Op. |
| Marshall, John Peter | Warren, O., | OALR |
| Marshall, Watson | 604 Diamond Bank Bldg., Pittsburgh, Pa., | ALR |
| Marquis, Geo. P. | 30 N. Michigan Blvd., Chicago, Ill., | ALR |
| Martin, H. H. | 247 Bull St., Savannah, Ga., | OALR |
| Martin, Wm. A. | 870 Fell St., San Francisco, Calif., | OALR |
| Mason, Albert B. | Waycross, Ga., | OALR |
| Masters, J. L. | 320 N. Meridian St., Indianapolis, Ind., | OALR |
| Matheny, Ralph E. | Galesburg, Ill., | OALR |
| Matheson, James P. | 511-513 Realty Bldg., Charlotte, N. C., | OALR▼ |
| Matthews, Justus | 1021 Metropolitan Bldg., Minneapolis, Minn., | LR |
| Mauldin, Leland Osgood | Greenville, S. C., | OALR |
| Maumenee, A. E. | 156 St. Frances St., Mobile, Ala., | OALR |
| Maurer, Joseph | Marion, Ind., | OALR |
| Maxey, Edward E. | 204 Idaho Bldg., Boise, Idaho, | OALR▼ |
| May, Chas. Henry | 698 Madison Ave., New York, N. Y., | Op. |
| May, James V. | 1701 Main St., Marinette, Wis., | OALR |
| Maybaum, Jacob E. | 17 E. 38th St., New York, N. Y., | ALR |
| Mayer, Emil | 40 E. 41st St., New York, N. Y., | ALR |
| McAllister, J. C. | Ridgeway, Pa., | OALR |
| McBride, Wm. Otis | 218 Washington Blvd., Fort Wayne, Ind., | OALR |
| McCaskey, Carl Heber | 3761 N. Illinois St., Indianapolis, Ind., | ALR |
| McClelland, Lefferts A. | 78 McDough St., Brooklyn, N. Y., | ALR |
| McCool, Joseph | 909 Stevens Bldg., Portland, Ore., | Op. |
| McCoy, John | 157 W. 73d St., New York, N. Y., | ALR |
| McCready, J. Homer | 816 Empire Bldg., Pittsburgh, Pa., | ALR |

McDiarmid, Henry O.....Brandon, Manitoba, Canada
 McDowell, Nathan Davis.....355 East Ave., Rochester, N. Y., OALR
 McGuire, Hunter H.....Winchester, Va., OALR
 McHenry, Dolph D...301 Colcord Bldg., Oklahoma City, Okla., OALR
 McKee, Claude W.....Greensburg, Pa. ▼
 McKee, Hanford.....158 Crescent St., Montreal, Canada. Op.A
 McKeenam, James W.....Washington, Pa.
 McKelvey, Alex. D.....193 Bloor St., E. Toronto, Canada
 McKeown, Elmer E.....708 Metropolitan Bldg., Denver, Colo., OALR
 McKernon, J. F.....62 W. 52d St., New York, N. Y., AL ▼
 McKinney, Richmond...Memphis Trust Bldg., Memphis, Tenn., ALR
 McLemore, Tipton.....Nevada, Mo., OALR
 McMurray, John B..Washington Trust Bldg., Washington, Pa., OALR ▼
 McNaught, Harvard Y....Butler Bldg., San Francisco, Calif., ALR
 McReynolds, Geo. S.....Temple, Texas, OALR
 McReynolds, J. O.....Trust Bldg., Dallas, Texas, OALR
 Means, Charles S.....131 E. State St., Columbus, O., OALR
 Meierhof, Edward L....1140 Madison Ave., New York, N. Y., OALR
 Mercer, Richard E.....608 Gas Office Bldg., Detroit, Mich., LR
 Merrill, Wm. Howe.....Lawrence, Mass., OALR ▼
 Messinger, Harry Carleton...170 Broad St., Providence, R. I., Op.
 Metz, R. B.....Guardian Bldg., Cleveland, O., Op.
 Metzenbaum, Myron.....768 Rose Bldg., Cleveland, O., ALR
 Mial, L. L.....23 W. 36th St., New York, N. Y., OALR
 Michie, W. T.....Scimitar Bldg., Memphis, Tenn., OALR
 Middleton, Alonzo Bolen.....Pontiac, Ill., OALR ▼
 Miller, Charles E.....Muncie, Ind., OALR
 Miller, Clifton M.....217 E. Grace St., Richmond, Va., OALR
 Millette, John W.....210 Reibold Bldg., Dayton, O., OALR
 Miner, S. G.....58 Cadillac Sq., Detroit, Mich., ALR
 Minor, Chas. L.....Fairbanks Bldg., Springfield, O., OALR
 Minor, Harry F.....Memphis, Tenn., OALR
 Mitchell, Edw. K.....704 W. Lehigh Ave., Philadelphia, Pa.
 Mitchell, Frederick.....Houlton, Me., OALR
 Mithoefer, Wm.....Lancaster Bldg., Cincinnati, O., ALR
 Mittendorf, A. D.....140 Madison Ave., New York, N. Y., Op.
 Mittendorf, Wm. F.....140 Madison Ave., New York, N. Y., Op.A
 Molt, Wm. F...529 Newton Claypool Bldg., Indianapolis, Ind., ALR
 Monaghan, Daniel G...564 Metropolitan Bldg., Denver, Colo., Op.
 Monahan, John Ambrose...181 Chestnut St., Clinton, Mass., OALR
 Mongel, Ernest.....1429 Tioga St., Philadelphia, Pa., Op.
 Monosmith, O. B.....424 Broadway, Lorain, O., OALR
 Monson, S. H.....328 Anisfield Bldg., Cleveland, O., OALR
 Moore, Fontaine Bruce....Exchange Bldg., Memphis, Tenn., OALR
 Moore, George A.....Bank Bldg., Palmer, Mass., OALR
 Moore, Mead.....The Rochambeau, Washington, D. C., ALR
 Moore, T. W.....Huntington, W. Va., OALR
 Moran, Timothy J.....702 Jenkins Bldg., Pittsburgh, Pa.
 Morgan, J. A.....Honolulu, Hawaii
 Morrison, Frank.....224 N. Meridian St., Indianapolis, Ind., Op.
 Mortimer, W. Golden.....40 E. 41st St., New York, N. Y., OALR
 Morton, J. P.....James St., Hamilton, Ont., OALR
 Mosher, Harris Peyton....828 Beacon St., Boston, Mass., ALR ▼
 Moss, Robert E.....Hicks Bldg., San Antonio, Tex., OALR
 Mott, John S.....1225 Rialto Bldg., Kansas City, Mo., OALR
 Moulton, Herbert...205 Merchants' Bank Bldg., Fort Smith, Ark., OALR
 Muckleston, Harold S....167 Stanley St., Montreal, Canada, ALR
 Mullin, W. V.....317 Barnes Bldg., Colorado Springs, Colo., ALR
 Muncaster, Stewart....907, 16th St., N. W., Washington, D. C., Op.A
 Muncy, Wm. M.....23 Waterman St., Providence, R. I., OALR
 Mundt, G. H.....25 E. Washington St., Chicago, Ill., OALR
 Munson, Dunham O.....Globe Bldg., Pittsburgh, Kan.
 Murdock, J. Floyd.....Bessemer Bldg., Pittsburgh, Pa.

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| Murphy, Frank G..... | Mason City, Ia., | ▼ |
| Murphy, J. W..... | 2711 Union Central Life Bldg., Cincinnati, O., | OALR |
| Murphy, W. E..... | 628 Elm St., Cincinnati, O., | OALR |
| Murray, W. R..... |519 Syndicate Bldg., Nicollet Ave., Minneapolis, Minn., | OALR |
| Nadeau, Emile G.... | Bellin-Buchanan Bldg., Green Bay, Wis., | OALR |
| Naftzger, Jesse B..... | 401 Davidson Bldg., Iron City, Ia., | |
| Nance, Willis O..... | 30 N. Michigan Blvd., Chicago, Ill., | OALR |
| Neeper, Edw. R..... | Box 567, Colorado Springs, Colo. | |
| Nelson, Louis Allan..... | 734 Lowry Bldg., St. Paul, Minn., | Op.A ▼ |
| Newell, Wm. S..... | 1424 K St., Washington, D. C. | |
| Newcomb, John Ray... | Hume-Mansur Bldg., Indianapolis, Ind., | Op. ▼ |
| Newhart, Horace.... | 910 Donaldson Bldg., Minneapolis, Minn., | OALR |
| Noble, Wm. Lincoln..... | 32 N. State St., Chicago, Ill., | OALR▼ |
| Norris, Samuel C..... | 16 W. 10th St., Anderson, Ind., | OALR |
| Norton, Charles E..... | 118 Lisbon St., Lewiston, Me., | Op.A |
| Norton, Daniel C..... | 967 Elm St., Manchester, N. H., | OALR |
| Norton, Roy R..... | Masonic Bldg., New Brighton, Pa. | |
| Noyes, Guy L..... |Columbia, Mo., | OALR |
| Noyes, Margaret L..... | 103 St. James Ave., Boston, Mass., | OALR |
| Nutter, Charles F..... | 16 Amherst St., Nashua, N. H., | Op. |
| Nye, F. T..... |Beloit, Wis., | OALR |
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| O'Brien, Steve A..... |Mason City, Ia., | OALR▼ |
| O'Connor, R. P..... |Oakland, Calif. | |
| Odell, Anna..... | 32 Adams St., Detroit, Mich., | Op.AL |
| O'Gwynn, J. C..... | 303 Van Ant Bldg., Mobile, Ala. | |
| Oertel, Theodore E..... |Augusta, Ga., | OALR▼ |
| Offut, W. N..... | Trust Co. Bldg., Lexington, Ky., | OALR |
| Oppenheimer, Seymour..... | 45 E. 60th St., New York, N. Y., | OALR |
| Orcut, Dwight C..... | 7 W. Madison St., Chicago, Ill. | |
| O'Reilly, Charles A..... | 1806 Chestnut St., Philadelphia, Pa., | ALR ▼ |
| O'Reilly, Wm. F..... |Lynn, Mass., | OALR |
| Orendorff, Otis..... |Cañon City, Colo. | |
| Orton, Henry B..... | 671 Broad St., Newark, N. J. | |
| Outwater, Samuel..... | 8th and Main Sts., Riverside, Calif., | OALR |
| Overman, Frederick Vaile..... | | |
| | 224 N. Meridian St., Indianapolis, Ind., | ALR |
| Owen, F. S..... | Brandeis Bldg., Omaha, Neb., | OALR |
| Page, Lafayette..... | Hume-Mansur Bldg., Indianapolis, Ind. | ▼ |
| Painter, Albin M..... | 512 Dollar Bank Bldg., Youngstown, O., | ALR ▼ |
| Park, John Walter..... | 32 N. 2d St., Harrisburg, Pa., | OALR |
| Parker, Elmer H..... | 820 Donaldson Bldg., Minneapolis, Minn., | LR |
| Parker, Walter..... | 32 Adams St., Detroit, Mich., | Op. ▼ |
| Parsons, Joseph Greeley..... |Sioux Falls, S. D., | OALR |
| Pattee, James J..... | 512 First Natl. Bank Bldg., Pueblo, Colo., | OALR |
| Patterson, James A..... | 214 Burns Bldg., Colorado Springs, Colo., | OALR |
| Patton, James M..... | 567 Brandeis Bldg., Omaha, Neb., | OALR |
| Payne, A. L..... |Eau Claire, Wis., | OALR |
| Payne, S. M..... | 542 Fifth Ave., New York, N. Y., | OALR |
| Pearson, W. W..... | 417 Equitable Bldg., Des Moines, Ia., | OALR▼ |
| Pendleton, F. M..... | 5th and Hampshire Sts., Quincy, Ill., | OALR |
| Peter, Luther C..... | 1527 Spruce St., Philadelphia, Pa., | Op. |
| Peterman, Harry E..... | 114 W. Franklin St., Baltimore, Md., | OALR |
| Pfafflin, Chas. A..... | Newton Claypool Bldg., Indianapolis, Ind., | OALR |
| Pfingst, Adolph O..... | Atherton Bldg., Louisville, Ky., | OALR |
| Pfingsten, Christian F..... | 508 N. Grand Ave., St. Louis, Mo., | ALR |
| Pfister, Franz..... | Majestic Bldg., Milwaukee, Wis., | ALR |
| Phillips, W. C..... | 40 W. 47th St., New York, N. Y., | ALR |
| Phillips, William L..... | 449 Franklin St., Buffalo, N. Y., | Op. |
| Phinney, Frank D..... | 22 W. 7th St., Cincinnati, O., | OALR |

- Pifer, John Daniel.....Joplin, Mo., OALR
 Pitkin, Carols E.....1021 Prospect Ave., Cleveland, O.
 Pollock, Harry Louis.....108 N. State St., Chicago, Ill., ALR
 Pontius, Paul J.....Professional Bldg., Philadelphia, Pa., OALR
 Porter, Edward H.....St. Madison St., Tiffin, O., OALR
 Porter, Lewis B.....277 Benefit St., Providence, R. I., OALR▼
 Posey, Wm. C.....1835 Chestnut St., Philadelphia, Pa., Op.
 Potter, Lester Forest.....278 Union St., New Bedford, Mass., OALR
 Potter, Peter.....411 Hennesy Bldg., Butte, Mont., OALR
 Potts, John Beakman.....567 Brandeis Bldg., Omaha, Neb., AL
 Powers, Everett.....Carthage, Mo., OALR
 Pratt, Fred J., Jr.....328 Central Ave., Minneapolis, Minn., OALR
 Pratt, John Abraham.....232 Coulter Block, Aurora, Ill., OALR
 Price, John W.....Bank of Commerce Bldg., Memphis, Tenn., OALR
 Price, Norman W.....Niagara Falls, N. Y., OALR
 Prout, Andrew Walter.....239 E. Town St., Columbus, O., OALR
 Putnam, Frank I.....Sioux Falls, S. D., OALR
 Pyfer, Howard.....Norristown, Pa., OALR
 Pyle, Wallace.....612 Bergen Ave., Jersey City, N. J., OALR
 Quittner, Samuel S.....5512 Woodland Ave., Cleveland, O., OALR
 Ralls, Clayton T.....Winfield, Kan., OALR
 Ralston, W. Wallace.....Scanlan Bldg., Houston, Tex.
 Ranly, John.....936 Clark St., Cincinnati, O., OALR
 Ravdin, Marcus.....223½ Main St., Evansville, Ind., OALR
 Ray, J. M.....300 Atherton Bldg., Louisville, Ky., OALR*
 Ray, Victor.....7th and Race Sts., Groton Bldg., Cincinnati, O., OALR
 Rebeck, Charles S.....412 N. 3d St., Harrisburg, Pa., OALR
 Rector, Albert E.....787 College Ave., Appleton, Wis., OALR
 Reeder, James E.....Sioux City, Ia., OALR▼
 Reaves, Wm. Perry.....Greensboro, N. C., OALR
 Reeve, A. R.....48 Bloor St., Toronto, Canada, OALR
 Reese, R. G.....50 W. 52d St., New York, N. Y., Op.
 Reger, H. S. 420-421-422 New Wellman Bldg., Jamestown, N. Y., OALR
 Reiche, Otto Carl.....Hazleton, Pa. ▼
 Remmen, Nils.....31 N. State St., Chicago, Ill., Op.
 Renaud, Geo. L.....Fine Arts Bldg., Detroit, Mich., OALR
 Renner, W. S.....341 Linwood Ave., Buffalo, N. Y., ALR
 Reyling, Fred. T.....1004 Oak St., Kansas City, Mo.
 Reynolds, H. G.....City Natl. Bank Bldg., Paducah, Ky., Op.A
 Rice, Geo. B.....293 Commonwealth Ave., Boston, Mass.
 Rice, Rodolph H.....Coshwell Bldg., Milwaukee, Wis., Op.AL
 Richards, Geo. Lyman.....124 Franklin St., Fall River, Mass., ALR
 Richardson, Chas. W.....
1317 Connecticut Ave., Washington, D. C., ALR ▼
 Richardson, James J.....
1509 Avenue of Presidents, Washington, D. C., ALR ▼
 Richardson, Robert M. 116 Fairview Ave., Chattanooga, Tenn., OALR
 Richie, Lee T.....106 E. Main St., Trinidad, Colo.
 Rideout, W. J.....Freeport, Ill., OALR▼
 Ridpath, Robert F.....2032 Chestnut St., Philadelphia, Pa., ALR
 Ringeburg, Eugene.....Lockport, N. Y., Op.
 Ringle, Chas. A.....Greeley, Colo., ALR
 Roberts, W. H.....461 E. Colorado St., Pasadena, Calif., OALR▼
 Roberts, Sam Earl.....703 Waldheim Bldg., Kansas City, Mo., ALR ▼
 Robertson, Chas. M.....32 N. State St., Chicago, Ill., OALR▼
 Robertson, Edwin N.....Concordia, Kan.
 Robertson, F. D. 724 The Ashton Bldg., Grand Rapids, Mich., OALR
 Robinson, John R.....Colorado Springs, Colo.
 Rogers, A. S.....105 S. Jefferson Ave., Saginaw, Mich., OALR
 Rogers, Benj. F.....222 Franklin St., Buffalo, N. Y., Op.A

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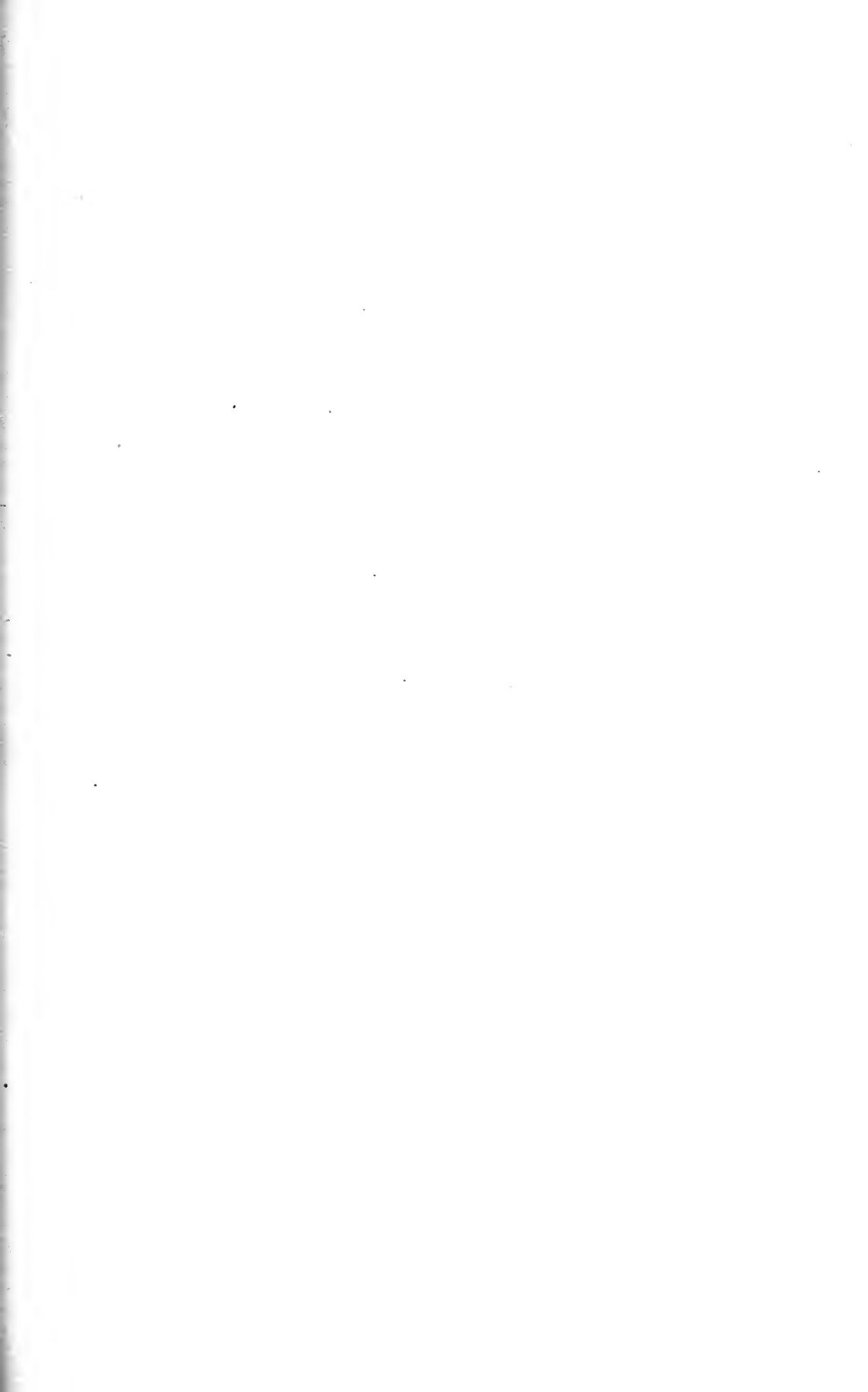
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| Rogers, W. K..... | 188 E. State St., Columbus, O., | OALR |
| Roller, Lewis A..... | 122 Monroe St., Grand Rapids, Mich., | OALR |
| Roost, Frederick H..... | Sioux City, Ia., | OALR▼ |
| Rott, Otto Mathias..... | 404 Paulsen Bldg., Spokane, Wash., | OALR |
| Row, George Samuel..... | 720-722 Indiana Pythian Bldg., Indianapolis, Ind., | Op. |
| Rowe, Ernest Harrison..... | 508 Park Ave., Baltimore, Md., | Op. |
| Rowland, John Franklin..... | Hot Springs; Ark., | OALR |
| Rubendall, Clarence..... | Brandeis Bldg., Omaha, Neb., | OALR |
| Ruby, F. M..... | Union City, Ind., | OALR▼ |
| Rucker, Edmond W J..... | 926 Woodward Bldg., Birmingham, Ala., | ALR |
| Russell, Edgar Reid..... | Legol Bldg., Asheville, N. C., | OALR |
| Rust, E. G..... | 316 Lennox Bldg., Cleveland, O., | Op.A |
| Ryan, L. R..... | San Marcos Bldg., Santa Barbara, Calif. | |
| Ryder, Delano Richmond..... | 124 Franklin St., Fall River, Mass., | ALR |
| Sanders, A. F..... | Groton Bldg., Cincinnati, O., | Op.A |
| Sanderson, David D..... | Lincoln, Neb. | |
| Sanderson, O. H..... | Washington Arcade, Detroit, Mich., | OALR |
| Satterlee, R. H..... | 187 Delaware Ave., Buffalo, N. Y., | Op. |
| Sauer, W. E..... | Humboldt Bldg., St. Louis, Mo., | ALR ▼ |
| Sauer, William W..... | Box 624, Marietta, O., | OALR▼ |
| Savage, G. C..... | 137, 8th Ave. N., Nashville, Tenn., | Op. |
| Savage, Geo. H..... | 1012 Central Bank Bldg., Memphis, Tenn., | OALR |
| Sawtell J. E..... | Waldheim Bldg., Kansas City, Mo., | ALR |
| Scales, John L..... | Shreveport, La., | OALR |
| Scales, J. W..... | Pine Bluff, Ark., | OALR▼ |
| Schaeffer, Geo. C..... | 112 E. Broad St., Columbus, O., | OALR▼ |
| Schappert, N. Louis..... | 57 S. Washington St., Wilkes-Barre, Pa., | OALR |
| Schenck, W. Edwards..... | 19 W. 7th St., Cincinnati, O., | OALR |
| Schild, E. H..... | Cleveland Ave and 9th St., Canton, O., | Op.A |
| Schiller, A. Noah..... | 1855, 7th Ave., New York, N. Y., | ALR |
| Schlegel, Herman..... | Wausau, Wis., | OALR |
| Schindwein, Geo. Wm..... | 138 W. 9th St., Erie, Pa., | OALR▼ |
| Schneideman, Theodore B..... | Professional Bldg., Philadelphia, Pa. | |
| Schneider, Francis A..... | Columbus, Ga., | OALR |
| Schoenberg, Mark J..... | 108 E. 31st St., New York, N. Y., | Op. |
| Scholz, R. P..... | Metropolitan Bldg., St. Louis, Mo., | ALR |
| Schrant, J. H..... | Hutchinson, Kan., | OALR |
| Schweinitz, Geo. E. de..... | 1705 Walnut St., Philadelphia, Pa. | ▼ |
| Scott, Burt L..... | Waco, Tex., | OALR |
| Schwenk, P. N. K..... | 1417 N. Broad St., Philadelphia, Pa., | Op. |
| Searcy, Harvey Brown..... | Tuscaloosa, Ala., | OALR |
| Sears, Wm. H..... | Huntingdon, Pa., | OALR |
| Seay, D. E..... | Trust Bldg., Dallas, Tex., | OALR |
| Sedgwick, Otis White..... | Alva, Okla., | OALR |
| Sedwick, Wm. A..... | 364 Metropolitan Bldg., Denver, Colo., | OALR▼ |
| Seegman, Simon..... | 8124 Jenkins Arcade Bldg., Pittsburgh, Pa. | ▼ |
| Selfridge, G. L..... | 240 Stockton St., San Francisco, Calif., | ALR |
| Sewall, E. C..... | 135 Stockton St., San Francisco, Calif., | OALR |
| Seydell, Ernest M..... | 105 W. Douglas Ave., Wichita, Kan., | ALR ▼ |
| Shackelton, W..... | 310 Osborn Bldg., Cleveland, O., | Op. |
| Shafer, Joseph J..... | Louisville, Ky., | OALR |
| Shahan, W. E..... | Metropolitan Bldg., St. Louis, Mo. | |
| Shanahan, Timothy J..... | 419 Boylston St., Boston, Mass., | ALR |
| Shanklin, E. M..... | Hammond, Ind., | OALR |
| Shannon, Chas. E..... | 1633 Spruce St., Philadelphia, Pa., | Op. ▼ |
| Sharp, Walter Nevins..... | 712-15 Hume-Mansur Bldg., Indianapolis, Ind., | Op. |
| Shastid, T. H..... | Superior, Wis., | OALR |
| Shea, John J..... | Exchange Bldg., Memphis, Tenn., | OALR▼ |
| Shellman, J. L..... | 935 Lowry Bldg., St. Paul, Minn., | OALR |
| Sherman, A. E..... | 108 Main St., Aurora, Ill., | OALR▼ |

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| Sherman, Elbert S. | Wiss Bldg., Newark, N. J. | OALR |
| Sherman, H. G. | 736 Rose Bldg., Cleveland, O. | OALR |
| Shields, J. Melville | Grand Junction, Colo. | OALR▼ |
| Shoemaker, Samuel A. | 416 W. Washington St., Bluffton, Ind. | |
| Shoemaker, W. A. | Carleton Bldg., St. Louis, Mo. | Op. |
| Short, Zuber N. | Dugan-Stuart Bldg., Hot Springs, Ark. | OALR |
| Shorter, James H. | The Grand, Macon, Ga. | OALR |
| Shreve, Owen M. | 162 W. 8th St., Erie, Pa. | OALR |
| Shuman, Geo. H. | East End Trust Bldg., Pittsburgh, Pa. | ▼ |
| Shurly, B. R. | 32 W. Adams Ave., Detroit, Mich. | ALR ▼ |
| Shute, D. K. | 1719 De Sales St., Washington, D. C. | |
| Simonds, Otis F. | 922 Rose Bldg., Cleveland, O. | Op. |
| Simpson, W. L. | Memphis, Tenn. | OALR▼ |
| Singleton, E. M. | Marshalltown, Ia. | |
| Skillern, R. H. | 2032 Chestnut St., Philadelphia, Pa. | LR ▼ |
| Sleight, R. D. | Battle Creek, Mich. | OALR▼ |
| Slocum, George | Ann Arbor, Mich. | Op. |
| Sluder, Greenfield | 3542 Washington Ave., St. Louis, Mo. | ALR |
| Smith, Dorland | 836 Myrtle Ave., Bridgeport, Conn. | Op.A |
| Smith, E. Terry | 36 Pearl St., Hartford, Conn. | OALR |
| Smith, F. K. | Warren, O. | OALR |
| Smith, Geo. Barker | Rome, Ga. | OALR▼ |
| Smith, Harry Austin | Delta, Colo. | OALR |
| Smith, H. E. | Norwich, N. Y. | OALR |
| Smith, J. F. | 86 Post St., San Francisco, Calif. | OALR |
| Smith, James M. | Valdosta, Ga. | OALR |
| Smith, Richard C. | Superior, Wis. | |
| Smith, Stanley S. | 613 Jenkins Bldg., Pittsburgh, Pa. | Op. ▼ |
| Smith, S. MacCuen | 1429 Spruce St., Philadelphia, Pa. | ALR |
| Smith, Victor C. | 1110 Maison Blanche, New Orleans, La. | Op. |
| Snow, Leslie W. | Templeton Bldg., Salt Lake City, Utah. | OALR |
| Snyder, F. D. | Ashtabula, O. | OALR |
| Snyder, Walter H. | 211 Ontario St., Toledo, O. | OALR |
| Sonnenschein, Robt. | Heyworth Bldg., Chicago, Ill. | ALR |
| Spalding, James A. | 627 Congress St., Portland, Me. | Op.A |
| Spencer, F. R. | Boulder, Colo. | OALR▼ |
| Spencer, Wm. H. | 1830 Rittenhouse Sq., Philadelphia, Pa. | |
| Spengler, John A. | Masonic Temple, Geneva, N. Y. | OALR |
| Spiece, Wm. Koenig | 1305, 29 E. Madison St., Chicago, Ill. | OALR |
| Spitze, Edward C. | East St. Louis, Ill. | Op. ▼ |
| Spohn, Geo. W. | Elkhart, Ind. | OALR |
| Sprague, Russel B. | 514 Commonwealth Ave., Boston, Mass. | ALR |
| Spurck, Michael D. | Peoria, Ill. | OALR |
| Stanberry, Henry | 8-10 E. 8th St., Cincinnati, O. | Op.AR▼ |
| Standish, Myles | 6 St. James Ave., Boston, Mass. | Op. |
| Stanford, James B. | Exchange Bldg., Memphis, Tenn. | OALR▼ |
| Stark, H. H. | El Paso, Tex. | OALR▼ |
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| Stauffer, Frederick | 164 E. So. Temple St., Salt Lake City, Utah. | OALR |
| Steim, Joseph M. | New Kensington, Pa. | Op. |
| Stein, Otto J. | 77 E. Washington St., Chicago, Ill. | ALR |
| Steuber, F. G. | Main and North Sts., Lima, O. | OALR |
| Stevens, Geo. T. | 350 W. 88th St., New York, N. Y. | Op. |
| Stevens, Henry B. | 410 Boylston St., Boston, Mass. | Op. ▼ |
| Stevenson, David W. | Akron, O. | |
| Stevenson, Robert | 22 W. 7th St., Cincinnati, O. | ALR |
| Stevenson, W. D. | 203 Majestic Bldg., Quincy, Ill. | OALR▼ |
| Stewart, Chas. C. | Auburn, Ind. | |
| Stieren, Edward | Union Arcade Bldg., Pittsburgh, Pa. | Op. |
| Stiles, C. M. | 1831 Chestnut St., Philadelphia, Pa. | ▼ |
| Stillson, Hamilton | 515 No. Bank Bldg., Seattle, Wash. | OALR |
| Stilwell, Hiram R. | 820 Metropolitan Bldg., Denver, Colo. | Op. |
| Stockler, Joseph A. | 228 N. 5th St., Reading, Pa. | |

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| Stoll, K. L. | 19 W. 7th St., The Vindonissa Bldg., Cincinnati, O., | OALR |
| Stotter, James | 352 Lennox Bldg., Cleveland, O., | OALR |
| Stout, G. C. | 1611 Walnut St., Philadelphia, Pa., | AL |
| Strader, Geo. L. | Cheyenne, Wyo., | OALR |
| Strong, James C. | Leadville, Colo., | OALR |
| Stricker, Louis | The Groton, Cincinnati, O., | Op. |
| Strickler, David A. | 615 Empire Bldg., Denver, Colo., | OALR |
| Strouse, A. N. | 79 W. 50th St., New York, N. Y., | OALR |
| Strout, Eugene S. | 2838 James Ave., South Minneapolis, Minn., | OALR |
| Stuart, Chas. C. | 504 New England Bldg., Cleveland, O., | Op. |
| Stucky, J. A. | 504 Fayette Natl. Bank Bldg., Lexington, Ky., | OALR |
| Stucky, Wm. S. | McClelland Bldg., Lexington, Ky., | OALR |
| Stueber, Paul J. | 209 W. North St., Lima, O., | |
| Sturges, Leigh F. | 128 W. 87th St., New York, N. Y. | |
| Sturm, Samuel A. | 5118 Jenkins Arcade Bldg., Pittsburgh, Pa. | |
| Suffa, George A. | 220 Clarendon St., Boston, Mass., | Op. |
| Suker, E. F. | 25 E. Washington St., Chicago, Ill., | Op. ▼ |
| Sulzer, G. A. | 237 E. State St., Columbus, O., | OALR |
| Sutphen, T. Y. | 997 Broad St., Newark, N. J., | Op. |
| Swan, Charles J. | 15 E. Washington St., Chicago, Ill., | OALR ▼ |
| Sweet, Wm. M. | 1205 Spruce St., Philadelphia, Pa., | Op. ▼ |
| Swetman, Charles R. K. | | |
| | 514 Consolidated Realty Bldg., Los Angeles, Calif., | ALR |
| Szymanski, Julius | Curitiba, Panama, Brazil, | OALR |
| Taber, Martin | Dallas, Tex., | OALR |
| Tangeman, Chas. W. | 607 Union Central Bldg., Cincinnati, O., | Op. |
| Tarun, William | 613 Park Ave., Baltimore, Md., | Op. A |
| Taylor, L. H. | 83 S. Franklin St., Wilkes-Barre, Pa., | OALR |
| Taylor, Lillian E. | | |
| | 1425 Marshall Field Annex Bldg., Chicago, Ill., | ALR |
| Taylor, Martin | 415 Warrington Ave., Pittsburgh, Pa. | |
| Teague, Rufus J. | Durham, N. C. | |
| Teal, F. F. | Lincoln, Neb., | OALR |
| Tenney, John A. | 419 Boylston St., Boston, Mass., | Op. |
| Thigpen, Charles A. | 13 Perry St., Montgomery, Ala., | OALR ▼ |
| Thomas, Charles D. | 705 Central Natl. Bank Bldg., Peoria, Ill., | OALR |
| Thomas, Frank W. | 331 Orr-Flesh Bldg., Piqua, O., | OALR ▼ |
| Thomason, H. E. | 310 Argyle Bldg., Kansas City, Mo., | OALR |
| Thomasson, Wm. J. | 942 York St., Newport, Ky., | OALR |
| Thompson, E. H. | 19 W. 7th St., Cincinnati, O. | |
| Thompson, Geo. H. | 18 Ashland St., North Adams, Mass., | OALR |
| Thompson, Henry Merrill | Pueblo, Colo., | Op. |
| Thompson, Peter Hunter | | |
| | 308 Commonwealth Ave., Boston, Mass., | Op. |
| Thomson, J. J. | 40 W. 47th St., New York, N. Y., | OALR |
| Timberman, Andrew | 112 E. Broad St., Columbus, O. | |
| Tindolph, Leah F. | Olean, N. Y., | ALR |
| Tingley, Louisa Paine | 9 Massachusetts Ave., Boston, Mass., | Op. |
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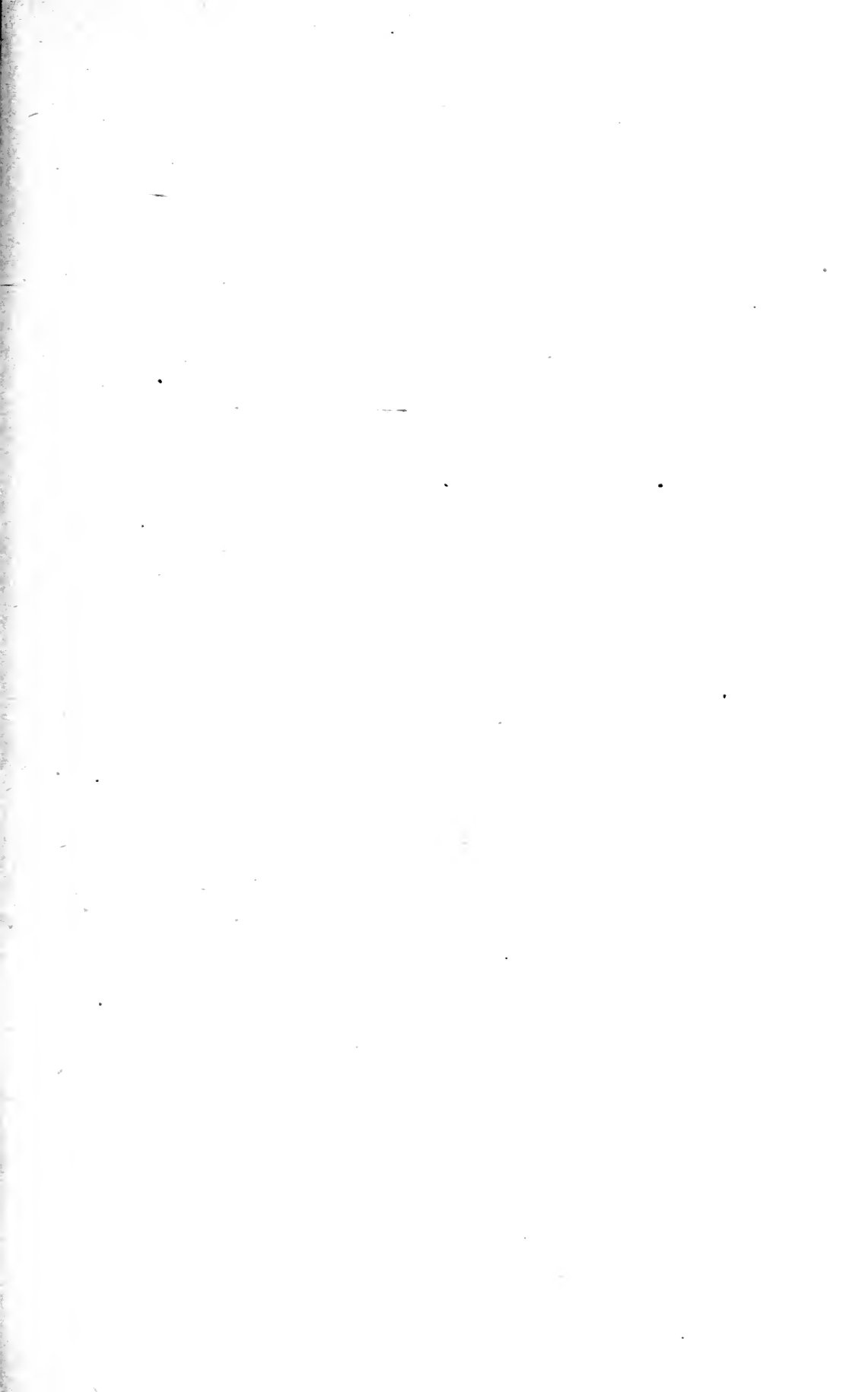
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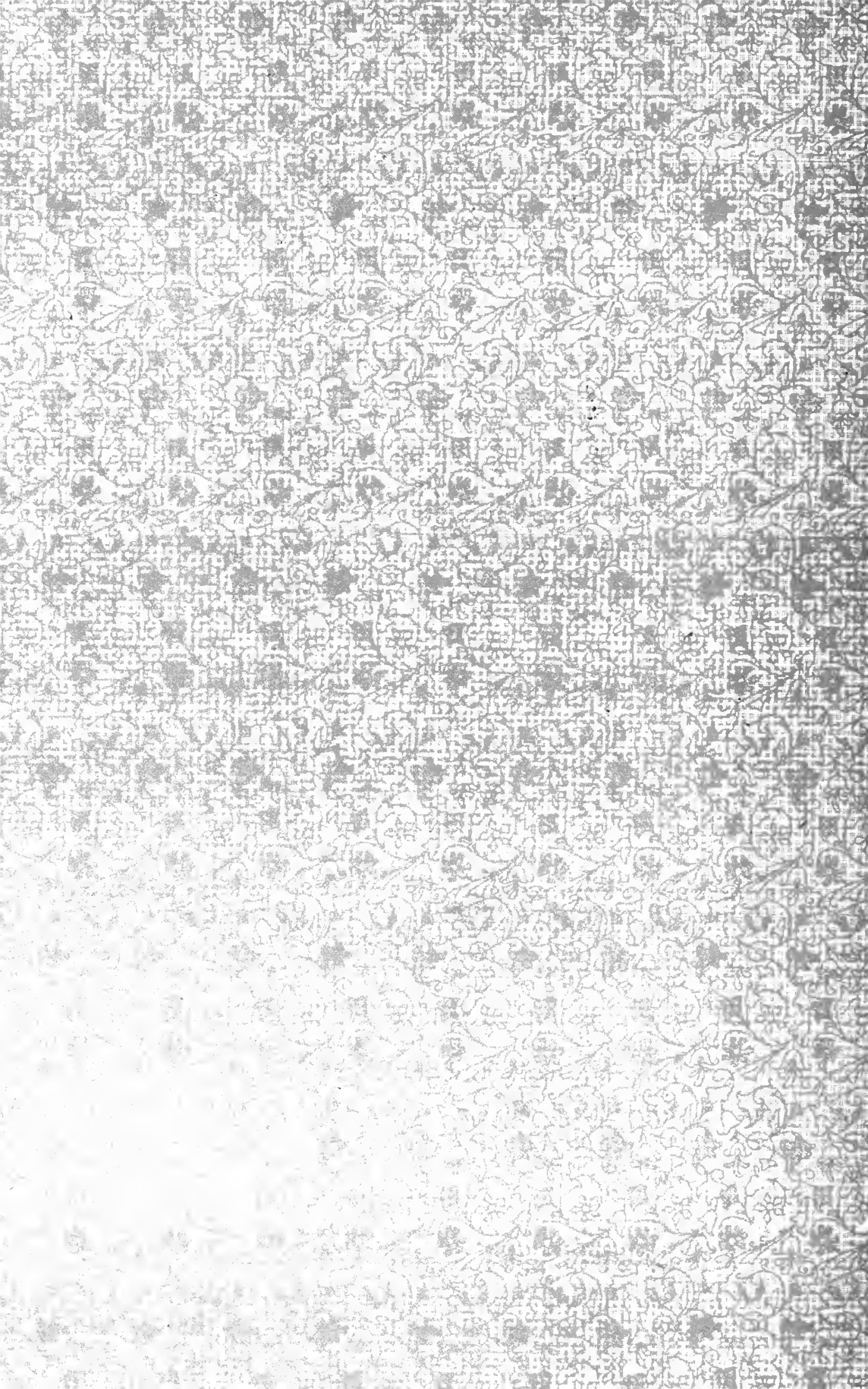
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